

# ARIZONA MEDICINE

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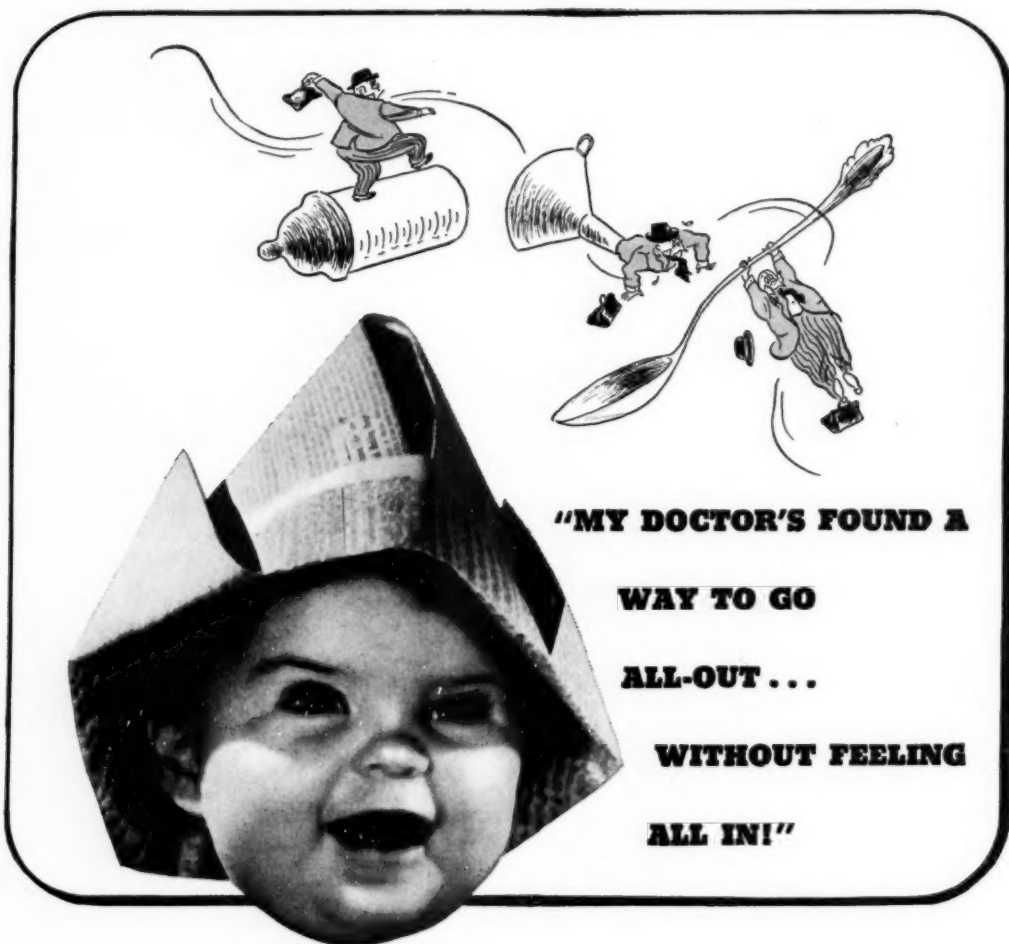
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**"MY DOCTOR'S FOUND A  
WAY TO GO  
ALL-OUT . . .  
WITHOUT FEELING  
ALL IN!"**

**"M**Y DOCTOR certainly hated figuring and re-figuring proportions of milk, carbohydrates, water for feeding formulas.

"Then he looked into S-M-A. And I was on S-M-A—as soon as he saw what a dependable way it was to shortcut that old arithmetic. In only two minutes he explained to my Mummy how to mix and feed my S-M-A.

*"He knows that in S-M-A I'm getting an infant food that closely resembles breast milk in digestibility and nutritional completeness.*

"Since my doctor put me on S-M-A I'm happy, strong 'n' growin'. Mummy's happy 'cause I'm happy, and feeding's easier for her. And Doctor's happy—'cause he can lick his extra wartime work without feeling all in.

"If you ask me—EVERYBODY'S happy if it's an S-M-A baby!"

• • •

Anutritional product of the S.M.A. Corporation,  
Division WYETH Incorporated

S-M-A is derived from tuberculin-tested cows' milk, the fat of which is replaced by animal and vegetable fats, including biologically tested cod liver oil, with milk sugar and potassium chloride added, altogether forming an antirachitic food. When diluted according to directions, S-M-A is essentially similar to human milk in percentages of protein, fat, carbohydrate, ash in chemical constants of fat and physical properties.

*Everybody's* **HAPPY IF IT'S AN** **SMA** **BABY!**

REG. U. S. PAT. OFF.

## PRIMARY CARCINOMA OF FALLOPIAN TUBE

MAURICE ROSENTHAL, M. D. and KENNETH PETERSON, M. D.

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**S**TUDY of the literature reveals that primary carcinoma of the fallopian tube is a comparatively rare lesion. In 1927, Barrows found only three primary cancers of the fallopian tube in 30,000 gynecological cases in Bellevue Hospital in New York City. Wharton and Kroek, in 1929, found only five cases in 35,000 gynecological patients in the Johns Hopkins Hospital. There is a discrepancy in the literature concerning the first authentic description of this condition. Ewing attributes the first description of primary carcinoma of the fallopian tube to Orthmann, in 1886. Nürnberg, in 1932, collected 301 cases covering the period from 1886 to 1931, and ascribed priority to Orthmann (1886). In 1935, Robinson, who reviewed the literature and reported three additional cases, stated that primary carcinoma of the fallopian tube was recorded for the first time by Reynard in 1847, and the first pathological description was rendered by Rokitsky in 1861.

There also seems to be a discrepancy as to the number of authentic cases reported. Ewing, in his Fourth Edition of "Neoplastic Diseases", states that about 120 cases had been reported since Orthmann's original description. In 1935, Robinson reviewed the literature, and recorded 349 cases. Novak, in his 1941 edition of "Text on Gynecological and Obstetrical Pathology", states: "There are approximately 300 cases to be found in the literature". Let it suffice to say that primary carcinoma of the fallopian tube has been recognized and recorded for many years, and that it is a comparatively rare finding.

The literature further reveals that it occurs most frequently during the fourth and fifth decades. However, it has been encountered as early as the second, and late as the eighth decade. It is usually a unilateral lesion, although one-third of the cases showed a bilateral involvement.

The symptoms and signs are not characteristic, although in a number of cases a low abdominal pain, with yellow watery or bloody

vaginal discharges were noted. The pain is colicky and intermittent in character, and is sometimes relieved as a sudden gush of watery yellow or bloody fluid is expelled from the vagina. Latzko thought that the colicky, intermittent pains were due to an exaggerated peristalsis of the tubes in an effort to expel the contents. Stanca felt that as a result of degeneration and necrosis the tubal contents may become fluid or semi-diffuent, and the fallopian tube attempts to expel this material into the uterus and vagina.

Physical examination of the pelvis is of little help in establishing a preoperative diagnosis, since it only reveals a mass, which may closely simulate any variety of pathologic changes in the fallopian tube. Furthermore, the diagnosis at the operating table is difficult to establish, as the tube is frequently enormously enlarged, and gives the impression of a pyosalpinx or hydrosalpinx. This is especially true, if it is associated with an intra-ligamentary or ovarian pathologic change. The external surface of the tube is smooth, and may present a purplish-blue discoloration.

Ewing states that the prognosis of tubal carcinoma is no better than cervical carcinoma, and after infiltration of the wall it is definitely unfavorable. Novak is also of the opinion that primary carcinoma of the tube is highly malignant, and found only a few reported cases which survived five years after operation. In 1926, Wechsler found only six 3-year survivals in 200 reported cases, according to Wharton.

### CASE HISTORY

The patient, a 45-year-old female, was admitted to the hospital following a periodic physical examination. Although she had lost 19 pounds in 9 months, the patient had no complaints, simply wishing an examination prior to returning to work.

The physical examination was essentially negative, except for a pelvic mass noted on the left side. Her past history was essentially negative. She had had no operations.

Her family history revealed that her moth-

er was living and well. Her father died at the age of 52, of heart disease. Her husband had pulmonary tuberculosis.

The patient started menstruating at 13 years of age, and was always regular. She had no miscarriages or abortions, and had been pregnant three times. She first became pregnant at 18 years of age, and again at 20 years of age, giving birth each time to a normal, full-term infant, with no difficulty. At 27 years of age she gave birth to full-term twins, both of whom died several hours after an apparently normal delivery. She did not know the cause of death.

The patient insisted that she never had any pain, and was merely undergoing a preliminary physical examination in anticipation of accepting work at a war plant.

At operation, an enlarged tube which resembled a pyosalpinx grossly was found. This tube, the other fallopian tube, with a small fibrous ovary, and the appendix were removed.

The gross pathologic description was as follows:

"The fallopian tube was markedly enlarged and distorted in shape. The tube was shaped somewhat like a retort. The external surface was quite smooth, and the fimbriated portion of the tube was completely obliterated. At this extremity the tube presented a large, nodular, cystic swelling. Upon opening the tube, the entire lumen was filled with a friable, grayish, granularlike mass of tissue divided by fine septa. The tubal wall appeared to be normal in thickness—in fact, in some areas it was thinner than usual. Upon sectioning the remainder of the tube it was also filled with a similar friable, grayish material which was hemorrhagic in some places. The other tube showed no gross changes of pathologic significance.

The ovary was small, oval in shape, and fibrous in appearance. It measured 1 x 2 cm. The appendix showed no gross changes of pathologic significance."

(However, microscopic examination revealed an acute inflammatory process of the appendix.)

Microscopic study of the enlarged fallopian tube revealed that the gray, bulky, friable mass described in the gross was comprised of large round epithelial cells arranged in an alveolar and papillary pattern. The stroma was scant, but moderately vascular, and was infiltrated by a moderate number of lymphocytes and plasma cells. The papillae or the papillary folds showed a hyperplasia of the

lining epithelial cells, possessing hyperchromatic nuclei and mitotic figures. There was only a slight tendency of the neoplasm to grow into the muscular wall, as only a few small gland spaces could be found in the sections studied. The fibromuscular coat in some areas showed atrophy of the muscular elements.

#### DISCUSSION

It is an interesting observation that primary carcinoma of the fallopian tube occurs so infrequently in contrast to the rather common occurrence of malignancy in other parts of the internal genital organs. This is especially interesting since the uterus, which is one of the commonest sites of malignancy, arises from the same embryonic structure—the muellerian ducts. Furthermore, there is a direct continuity between the uterus and the fallopian tube. However, as one examines the difference in histologic structure of the uterus and fallopian tube, one observes a difference in the function and structure, which may account for the frequency of malignancy in the uterus. In the uterus one finds three types of epithelium, in the endometrium, the cervical canal, and the surface of the external os; whereas in the tube the epithelium is uniform in structure and apparently only reacts slightly to hormonal stimulation produced by the ovary. In the uterus, the epithelium responds by hyperplasia, hypertrophy or atrophy to hormonal stimulation. While the fallopian tube has a simple function—merely that of transmitting the ovum to the uterus—the uterus has many functions, and is constantly undergoing various structural changes. Furthermore, the cervix and uterus are frequently exposed to traumatic and chronic irritations. This is especially true of the cervix. For example, repeated pregnancies may cause cervical tears, and other trauma of the cervical tissue. The fallopian tube, being in the pelvis, is protected from trauma. It is frequently irritated by infection, although not as frequently as the cervix. Since trauma and chronic irritation are usually accepted etiological factors in malignancy, it is therefore not unusual to find malignant changes in the more frequently traumatized and chronically irritated portion of the internal genital organs.



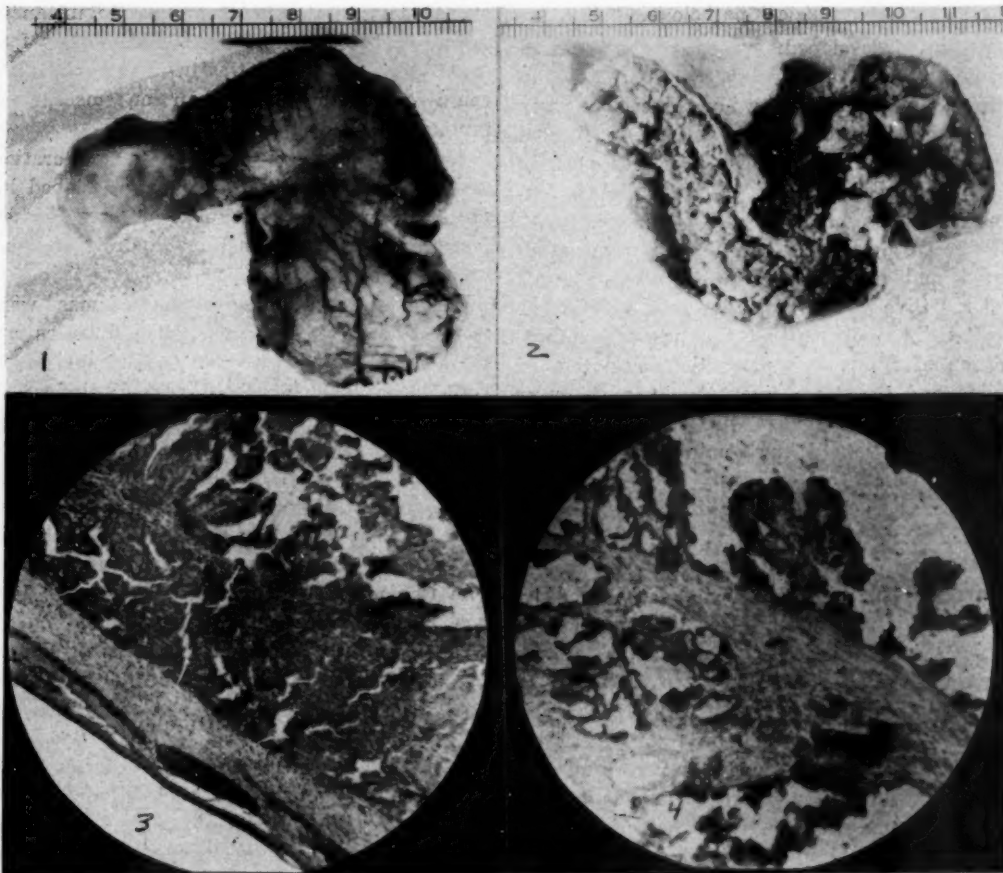
The tumors arise from the mucosa of the tube, which may produce a papillo-alveolar or solid architectural pattern. The tumor mass tends to grow into the lumen of the tube, and the wall of the tube usually remains thin. This is in contrast to metastatic carcinoma to the fallopian tube, in which the tumor often arises in the wall of the tube, producing a thick wall.

Secondary infection may occur, producing degeneration and necrosis of the tumor cells. The malignancy may spread into the peritoneal cavity by way of the open fimbriated end of the tube, producing peritoneal implantations with ascites and diffuse carcinomatosis. Occasionally, it spreads by direct invasion through the wall of the tube into the mesosalpinx, and

thus may involve the ovary, intestine, or uterus. Metastasis by way of the lymphatics to distant lymph glands may occur.

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1. Note the smooth external surface of the tube, and the resemblance to a pyosalpinx.
2. Cut surface of tube showing lumen filled with a grayish, granular tumor mass.
3. Note the papillary variety of carcinoma growing from the thin wall of the tube.
4. Large tree-like papillae growing toward the center of the lumen.

## THE BIOCHEMICAL APPROACH TO MODERN SURGICAL METHODS

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IN the treatment of many surgical conditions, we have witnessed the evolution of the education of a surgeon from that of the skilled anatomist to the combined and much preferred moderately well versed anatomist, and excellent surgical pathologist. In order to treat scientifically and with reason many surgical afflictions, the surgeon must incorporate into his working armamentarium a thorough knowledge of biochemistry as it is related to the allied specialties, and have the worthy attributes of his predecessors by being skilled in anatomy, physiology, and surgical pathology.

Recently developed surgeons have passed through a very much troubled and chaotic intern year with perhaps two or three more years of residence in some line of special surgical endeavor. A great deal of their daily work in the hospitals appeared to be just so much routine with little apparent reason for it at times. On the surgical wards, often they have anticipated and executed the attending surgeon's order, "Give her some fluids—that will fix her up," little realizing that the unwise administration of fluids as well as the dissipation of fluids and secretions was fraught with grave dangers. All of us have witnessed in the days of popular gastro-jejunostomies for peptic ulcer, the cases that suddenly turned for the worse on the eighth or tenth day of an uneventful convalescence after a perfectly executed technical job. The unsuspected late and fatal complications were usually followed by reopening the abdomen after thorough supersaturation of the patient with NaCl eyses, thereby adding further insult to a disordered G. I. tract, made so by a faulty electrolyte balance, and a distortion of serum protein levels altering the colloid osmotic pressure of the blood. By doing an anterior gastro-enterostomy, a procedure frequently performed, and by making a larger stoma with more strangulated tissue, resulting in greater suture edema,

and the further supersaturation of the patient with NaCl and with no thought of the protein carbohydrate and mineral balance necessary for maintaining colloid osmotic pressure of the blood, the patient remained inexplicably ill. The vicious circle increased, and we proposed suction as an adjuvant further depleting Cl ions leading to alkalosis. The edema around the newly made suture line often became worse with protracted vomiting. As an adjunct to such surgical therapeutics, surgeons resorted finally to jejunal feeding with all descriptions of peptonized solutions and in some instances a happy outcome supervened. To the less fortunate who died, there was little to explain the cause of death other than on mechanical grounds.

Little thought was given to the restoration of the plasma colloid pressure of the blood by the use of transfusion, of lyophilized serum, plasma amino acids, peptone hydrolysate and hypertonic solutions.

Procedures producing tissue edemas were ironically and persistently adhered to as acceptable therapy with an occasional blood transfusion to change the order of things. It is now a known fact that blood transfusion only temporarily restores circulating blood proteins to normal, after which the blood protein levels revert again to their former lowered state. Elman of St. Louis has demonstrated in his research that parenteral restoration of protein is alone insufficient; in that the mechanism for protein regeneration must be reactivated to restore permanently the serum protein level of the blood and prevent the disastrous train of symptoms resulting from protein depletion.

The factors producing tissue edemas after vigorous unphysiological NaCl administration produce the late and latent suture line edemas and cause breaking-through of suture lines, leaking, and resultant fatal peritonitis often wrongly attributed to soiling at the time of operation.

The problem of water intoxication of the

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(The thoughts expressed in this article are not to be construed as the policy of the armed forces but are strictly the private opinion of the author.)

surgical patient has been studied. Through painstaking efforts of Collier, Maddock, et al, who called attention to *water loss, water storage*, insensible water loss, and the fluid requirements of the "genus homo." Without this knowledge, many types of elective and emergency gastro-intestinal surgery would eventuate unnecessarily in a surgical mortality.

By placing the fluid requirements of man upon a scientific basis we are now able to gauge more accurately first and subsequent p. o. days' fluids requirements in "CC's" and to prevent such extreme states of either hyperhydration to the point of intoxication and its converse dehydration with its attendant acidotic features and extremes of tissue dessication.

As embryonic surgeons, we have heard "the chief" say, "Give her five thousand today; she has peritonitis—we must wash those poisons out through the kidneys and skin." Often, we found, to our chagrin, that as quickly as the fluids were poured into the circulation by venoclysis at a rate usually far too rapid for absorption, that these—with other threshold substances—were found in the jar along the side of the bed in a far larger amount than the original clinical order called for. This further augmented abdominal distension, decreasing urinary output, increasing nitrogen retention, increasing the already existent edema of visceral organs, and the loss of precious crystalloids so necessary for the maintenance of blood neutrality (P. H.).

The syndrome of hypochloremia is well established now, such as occurs in pyloric obstruction, fistulae, diarrheal states, and the like. The role that NaCl plays in certain so-called deficient glandular states as hypoadrenalinism and Addison disease must likewise be reckoned with in many postoperative surgical conditions. The relation of the cortical portion of the adrenal to the shock syndrome has given us an added avenue of approach to the treatment of shock.

Before Virgil Moon's investigations on shock, the etiological differences in the mechanism producing the shock syndrome as caused by hemorrhage, or by the release of histamine-like substances causing vasomotor paresis, or the shock supervening upon acute trauma were unappraised. It is gratifying to know that serial hemoglobin determinations reveal as

much information about the state of shock, its course and prognosis, as do serial blood pressure determinations with the usual clinical admonition following to give more fluids that readily filtered out of the circulation along with such stimulants, which, because of their central action, had no therapeutic effect upon the stasis of blood in the splanchnic cesspool and the peripheral vascular bed—namely, the vast capillary and arteriolar bed.

The classification of shock has led to a more concise, scientific and workable understanding of its therapy, and has extended therapeutics to the use of cortical extracts (hormonotherapy) and the use of crystalloids of proper molar concentrations, and blood plasma when indicated, and such peripheral vasomotor stimulants that have a direct action upon the blood vessels maintaining vasomotor tone, and the vasomotor center in the medulla.

The physiology of shock is slowly being completed due to the work of Scudder, Moon, Blalock, and a host of unsung physiologists. It certainly is clinically worthwhile to know whether the causative factor producing shock is due to loss of fluids, toxic absorption from a burned surface with its associated histamine-like action, trauma, or perhaps violent hemorrhage. The therapy of shock suggests itself to the careful clinician for it would be worthless to transfuse in a case where an already pathological polycythemia and lowered plasma volume of the blood existed. Likewise, it would be stupid to give isotonic solutions to the patient shocked from hemorrhage without the preliminary blood transfusion; or administration of acacia or plasma; or, as I have witnessed many a time, the giving of central stimulants such as digitalis, coramine, strophanthus when peripheral vasomotor collapse and arteriolar stasis existed.

I have seen, as have others, the use of fluids, morphine, and the administration of intravenous infusions abused to such an extent that I feel that every one qualified in surgery should be versed in the biochemical reasoning for the use of these substances with some scientific accuracy. An internist of good repute would no longer think of controlling diabetes without accurate quantitative and qualitative urine and blood determination. A surgeon,

however, will blithely disturb the water, electrolyte, and vitamin balance, and necessary blood protein levels to such an extent to literally shock the entire biochemical and biophysical makeup of the patient and continue upon his way after doing acceptable mechanical work. I know that it is possible in every surgeon's experience to cite cases of water intoxication, hypochloraemia, gastro-intestinal atonies and the like that righted themselves either because of the surgeon's aloofness or ignorance of what to do, or because of that grand stabilizing force and biological reserve of mother nature in the face of odds with the constant desire to right the wrongs of our well meant unphysiological endeavors.

The true understanding of the biochemical aspects of high intestinal obstruction with the significant diminution in the plasma chloride concentration and its attendant alteration in electrolyte distribution and acid base balance has saved many a patient from doom. Prolonged losses of gastric juices, bile, jejunal and ileal fluids through vomiting, diarrhea, or external fistulae result in depletion of blood chlorides and resultant hypochloraemia. The actual chloride loss in these conditions is masked by the associated states of dehydration and tissue desiccation such that the true index of chloride requirement of the body is often masked by plasma chloride concentration determination.<sup>7</sup>

In hypertrophic pyloric stenosis of infants and the stenosing duodenal ulcer of adults, chloride depletion is usually in excess of Na loss. The loss of chloride from the blood plasma results in the accumulation of  $\text{HCO}_3$  and if protracted, plasma sodium is depleted by its excessive elimination in the urine in an attempt on the part of the body to maintain blood neutrality (ph) and prevent the development of a masked alkalosis.<sup>7</sup>

Today we think in terms of alkalosis and its attendant symptoms. Alkalosis due to primary alkali excess is one of the most constant metabolic features of upper intestinal obstruction together with hypochloraemia, and nitrogen retention. As a result of the loss of the Cl ion from the blood there is present in the blood an excess of base chiefly Na and K retained in the form of  $\text{HCO}_3$ . A point worthy of mention is that ketosis can occur in the presence of alka-

losis as well as acidosis. Ketosis develops as the result of CHO starvation, but the acidifying effect of the ketone acids is insufficient to counterbalance the excessive  $\text{HCO}_3$  accumulation resulting directly from the depletion of the chloride ions. When adequate Na, Cl, and  $\text{H}_2\text{O}$  are administered, the correct degree of retention and excretion of the Na and Cl ions necessary to reestablish the normal acid base equilibrium depends upon kidney function and plasma protein levels. The ketone acids are corrected by glucose administration and insulin in proper dosage.

Scudder, Zweemer, and Whipple<sup>7</sup> in an evaluation of 2150 cases of intestinal obstruction revealed K to be the toxic factor.<sup>6</sup> The Mayo group in their research reveal the associated great losses of Na, Ca, and K in refutation of Scudder's work. We now have the solution containing the bases of K and Ca (Ringer's Solution) added to saline and dextrose. Falemer, Osterberg, and Eargen have demonstrated that high cholesterol values in high intestinal obstruction are usually of serious import.<sup>2</sup> More recently high value for K were found in intestinal obstruction—values as high as mg./100 in clinical and experimental obstruction. The increase was said to be due to the absorption of K from contents of obstructed bowel; others attributed it to adrenal cortical insufficiency.<sup>5</sup>

That certain of the inorganic constituents of the blood serum exist in ratios that can be determined clinically was brought out by Ellen S. Flurey<sup>3</sup> of the Mayo Clinic who determined that the mean value for total protein was 7.4 gm per one hundred cc., for albumin 4.9 gm and for globulin 2.4 gm.<sup>3</sup> The mean albumen globulin ratio was 2.06, the mean value for sodium 332.5 mg., for K 19.1 mg., and total base 159.2 cc. of tenth normal sodium hydroxide per 100 cc of blood. It was further brought out in this study that there is no significant variation in the mean values of the various groups. That the probably variation of mean values varied only from .02 to 1.52 per cent lends itself to studies on cases in which significant deviations indicate disease; that the surgeon today is forced to use these adjuncts in physical diagnosis goes without saying; not to use them, very often determines a poor clinical result ending disastrously.



Interesting cases of spontaneous hypoglycemia and their amelioration by resection of the islet cells of the Pancreas have come to the attention of surgeons. Blood sugar determinations and sugar tolerance curves and the finding of islet cell tumors has led to courageous attacks upon the pancreas. Today it is found that there are certain cases of unusual spontaneous hypoglycemia which do not yield to the classical Whipple approach and resection. This is no more disconcerting a fact than the knowledge that diabetes mellitus is not solely a pancreatic disease due to deficiency, sclerosis or atrophy in the Islet cells of Langerhans. More interesting biochemical speculation that will ultimately lead to a greatly enlightened approach to the problems of hypoglycemia as occasioned by Islet cell tumors of the pancreas, and the more accurate control of insulin resistant diabetes will eventuate.

In the field of biliary surgery, the hazardous case of obstructive jaundice with its coexistent effects upon blood coagulability have been rendered safer for operative intervention by the use of Vitamin K. Doctors Butt, Snell, and Osterberg<sup>1</sup> have demonstrated that the administration of vitamin concentrates containing Vitamin K along with human bile or bile salts resulted in a decrease of the prothrombin time of the blood of patients having jaundice. Actual bleeding is said to be inhibited by the administration of these products. The true chemical composition of Vitamin K in pure form will soon be known, but it is definitely known that a deficiency of this accessory fat soluble substance produces a bleeding tendency. The exact nature of prothrombin is uncertain. It is thought to be a protein and for the present the qualitative method of Quick and his co-workers is best adapted for clinical usage.<sup>4</sup> Just as naval surgeons worked out Vitamin C, so will Vitamin K be worked out in pure form. This we know: the administration of this vitamin (1) decreases mortality in operation on the biliary tract of obstructive jaundice with prolonged bleeding time, (2) decreases the incidence of abnormal post operative bleeding, and the necessity for blood transfusion. Just as the surgical diabetic is treated by group effort, so the surgically jaundiced patient, with the one admonition that we must not be lulled into a sense of false security, but must use all

means available today, plus the better way, and prevent the occurrence of bleeding and do not sub-judice refute some of the older tried methods.

Blood chemistry determinations are of particular value in the practice of internal medicine and surgery, and as such allow both the internist and surgeon to evaluate the patient as a surgical risk, thereby enabling the adoption of the best suited anesthetic agent to the surgical procedure after due preparation of the patient with regard to mineral, carbohydrate and vitamin balance, sugar storage in the liver, oxygen carrying capacity of the blood and its coagulability. Surgery is today no longer out of reach of the debilitated patient, and by the adequate use of biochemical studies, the one-time condemned patient can be restored to a state of operability by (1) augmenting kidney function, (2) increasing heart reserve, (3) restoring mineral and vitamin balance, and correcting blood, glandular, and vitamin deficiencies. Without the aid of the laboratory and biochemical methods, much of the preparation which a surgical case receives today would be lost in the quagmire of presupposition and unintelligent guessing. It is just as important to have the surgical patient in *mineral, water, and vitamin balance* as it to have the necessary amount of *sugar and protein* stored in the liver, as glycogen is to be used during anesthesia and convalescence. No less an instrument than the electrocardiograph often forestalls many a gall bladder operation by detecting organic disturbances of heart rhythm and heart muscle deficiencies that would go unrecognized by the usual methods of diagnosis. The relative acuteness, or chronicity of an inflammatory process is again appraised by the white blood count and differential studies, before surgery is attempted, such that the surgeon is somewhat informed of the fight the natural defenses are waging against an infection. An appraisal of the oxygen carrying capacity of the blood by routine hemoglobin determinations will often make a patient a better risk if blood transfusion is undertaken prior to surgery rather than after surgery. It is not only of scientific interest, but to the interest of the patient, to have some idea of the circulating proteins in the blood and the ratio in which these exist in the blood stream.



Only within the past few years has it been a known fact that deficiencies in the circulating blood proteins and in Vitamin C lead to disruption of wounds, delayed healing, and poor tissue regeneration. Only through biochemical studies have the surgeons avoided unnecessary waterlogging of the tissues after operation by their vigorous, well-meant attempts to support the circulation and restore electrolytes, little realizing that sodium and chlorine exist in a definite proportion in the blood plasma and are there to maintain proper osmotic relationships between the blood and the surrounding tissues. Certain types of non-mechanical upper intestinal obstruction due to deficiencies of proteins in the blood are rapidly corrected by the administration of one of the most potent blood proteins—namely, the matched blood of a suitable donor.

It is a rare sight to see a patient almost moribund, waterlogged, sluggish and just not getting along, suddenly become bright, snappy and with appetite restored after the doctor orders meat for the patient, following the interdiction of salt. Simple as numerous of these observations of a similar nature may appear, all are sanely and scientifically proven, and without methods of precision such surgical work would come needlessly to a bitter end. The preparation of patients having a tendency to hemorrhage—with Vitamin K to augment blood coagulability—is striking in the cases of

benign and malignant obstructive biliary disease. The preparation of these patients for safe surgical approach constitutes a problem for the combined efforts of the laboratory, internist, and surgeon. Here, through the use of Vitamin K, bile salts, glucose and protein, the coagulability of the blood is restored to normal; the small intestine digests fats better and the liver function is restored to a safer level of glycogen reserve. Thus, it is plain to see that surgeons of today must have graduated from the stage of "barbour surgeons" of the middle ages where only a skilled hand and stronger assistants were necessary to restrain a patient in the dorsal recumbent long enough for the surgeon to perform his work. Today, the surgeon must combine the services of anatomist, pathologist, and biochemist and craftsman who must work manually to aid nature in the physiological (biochemical) restoration of function to diseased organs.

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## DIAGNOSIS WITH DIFFERENTIAL DIAGNOSIS OF SILICOSIS FROM OTHER CONDITIONS SIMULATING IT

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IT is generally recognized that silicosis often is a disease slowly developing over a period of years and that in its early stages there may be no clinical symptoms. Watkins-Pitchford found that on the average it required 9.6 years before the appearance of symptoms following exposure. Smith claims that silicosis can only be reliably diagnosed by x-ray of the chest. She found that in 25% of a group of cases whose x-rays showed unmistakable evidence of silicosis there were no symptoms whatsoever

and that therefore symptoms, while significant when found, do not by any means indicate absence of silicosis by their absence. On the other hand the reverse may also be true for there are cases that develop cough and shortness of breath with only slight x-ray evidence of increase in the root shadows and on the accentuation of peribronchial markings as emphasized by Cooke.

The diagnosis of silicosis is not difficult in the advanced stages but it may be overlooked

in individuals who have not been exposed to dust for a period of years. Britton and Head cite four cases that were exposed to silica dust for from 4 months to 14 years without developing symptoms but that years later, after a prolonged interval in which they were unexposed to dust, developed symptoms and x-ray evidence of silicosis. From these cases it is seen that the lung changes may, and often do, continue long after the inhalation of dust has been discontinued, and therefore makes it important to obtain a careful occupational history in all cases in which the symptoms and x-ray findings suggest the possibility of silicosis.

There is an acute form of silicosis reported by Kilgore which is often overlooked and may be difficult to diagnose unless it is kept in mind. He cites 6 cases that were exposed to silica for from 10 to 16 months who developed acute symptoms of dyspnea and cough. A few also had chest pain and hemoptysis. An erroneous diagnosis was made in a few of these cases because of the rapid fatal outcome of their disease. All of these cases were in the same industry—the manufacture of scouring powder for domestic use. It is probable that the accelerated course of the silicosis in these cases was due to the combined inhalation of alkali or soap, or both, with quartz dust and that the mixture greatly increases the harmful effect of the silica.

Except for these two rather uncommon types of silicosis, the one in which the disease progresses long after exposure has ceased and the other acute form occurring in the scouring powder and soap industry, silicosis commonly runs a uniform course which, for matter of convenience, has been divided into three stages. The committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association has given the following statement in regard to the stages of the disease:

"First stage: (Corresponding to ante-primary stage of South Africa) The symptoms of uncomplicated first-stage silicosis are few and often indefinite. The man may apparently be quite well and his working capacity not noticeably impaired. Slight shortness of breath on exertion and some unproductive cough often with recurrent colds, are the most usual symptoms. The man may have a little less ability to expand his chest than formerly, and the elastic-

ity of the chest may be slightly impaired. The earliest specific indication of the presence of silicosis is the radiographic appearance consisting of generalized arborization throughout both lung fields with more or less small, discrete mottling."

Sayers states that no nationality is exempt from the disease and that all races are susceptible, and that age in itself is probably no great factor in the prevalence of the disease. Irvine claims that 0.83 per cent of miners in South Africa had silicosis at 30 to 35 years of age, 2.61 per cent at 34 to 37 years, 3.77 per cent at 42 to 45 years, and 3.5 per cent at over 46 years. On the other hand, in other reports the incidence has been much higher but there is a general agreement that the incidence rises with advancing years because of the increased length of exposure to the silica dust. It has been further systematically noted in all extensive reports of the disease that shortness of breath to a greater or less degree is a cardinal symptom of the first stage of silicosis. Irvine states that there is some amount of irritating cough, typically a deep cough with little or no expectoration often worst in the morning and sometimes inducing vomiting. According to Irvine, it is the belief of the investigators in South Africa that "it remains true to say that we should not think of 'simple' silicosis merely as a dust fibrosis but as being, at least in many cases, a dust fibrosis which from its beginning as a clinically detectable condition is linked up with an element of latent tuberculous infection."

Symptoms such as expectoration, hemoptysis, and night sweats were slightly increased in the first stage of silicosis but probably were indicative of infection. In summarizing, Irvine states that during the first stage of silicosis the following signs are usually present:

1. A certain lack of elasticity of the chest wall during movements of respiration together with
2. A somewhat reduced air entry and
3. A characteristic alteration of the inspiratory murmur from the normal "vesicular" character to a higher pitched or "harshened", "thinned", and commonly somewhat shortened type, the expiratory murmur, although prolonged, remaining fainter than the inspiratory.

Irvine further states "that this type of breath sound is very characteristic, with some

modification, of silicosis in all its stages. It is first noticeable at the anterior, lateral, and basal regions of the lungs. In a minority of cases, he finds, however, that the breath sounds may be simply diminished, usually no rales are heard."

In general there is an agreement in the literature with the signs and symptoms given by Irvine for the early stages of silicosis. On the other hand, it would be impossible to make a diagnosis of the condition on these meagre grounds without a careful history of exposure to the silica dust and without x-ray evidence of the disease.

In the second stage of silicosis, which corresponds to the primary stage of South Africa, a definite shortness of breath upon exertion is usually found, and pains in the chest are frequent complaint. A dry morning cough is frequently present, sometimes with vomiting and recurrent colds are frequent. Even then the man's appearance may be healthy but he is dyspneic on exertion, he cannot work as well as formerly and his chest expansion is noticeably decreased. The characteristic roentgenographic appearance is a generalized medium-sized mottling throughout both lung fields. The shadows of the individual nodules are for the most part discrete and well defined on a background of fibrous arborization, but there may be here and there larger but limited opacities due to pleural thickening, or to a localized aggregation of nodules.

It is remarkable how well men often are during this stage and how little they are physically handicapped to carry on their strenuous work in the mines.

In the third stage (corresponding to the secondary stage of South Africa) the shortness of breath is marked and distressing even on slight exertion. The symptoms enumerated for the second stage are present but more marked, the cough is more frequent, the expectoration is in most cases slight but may be copious. The individuals' capacity for work becomes seriously and permanently impaired; his chest expansion is greatly decreased, he may lose flesh, and there may be symptoms of cardiac decompensation. The radiographic appearances in the third stage are further accentuated, the mottling is more intense and the nodules are

larger and take on a conglomerate form so that the larger shadows are shown corresponding to areas of dense fibrosis.

Irvine states that few cases of advanced silicosis are without the infectious element although this may be latent and not apparent clinically. He noted that the general nutrition may be quite good and that many of the men are obese. With few exceptions there is some cyanosis and some obvious dyspnea. The chest has the appearance of moderate emphysema and a greater or less degree of emphysema is a constant feature, the chest expansion is decidedly impaired, the percussion note is dull to flat. Inspiration is short and decidedly reduced, and expiration is high pitched but may be merely reduced. Occasionally, secondary cardiac dilatation with marked cyanosis and edema, eventually resulting in death, are seen.

The more recent studies of silicosis in this country agree closely with the observations of Irvine who further calls attention to the fact that the supravention of active tuberculosis is usually very clearly indicated by a definite and progressive loss of weight, increased cough and expectoration, sometimes with hemoptysis and rapid breakdown of the patient's condition. Dyspnea appears to be out of proportion to the actual extent of infection. Sayer's reports that 267 cases of silicosis plus tuberculosis were found among the 7,722 men examined in the Picher group and the observations in these cases were similar to those described by Irvine.

From the many studies reported in the literature it may be concluded that the cardinal symptoms of silicosis is shortness of breath, the cardinal sign decreased chest expansion, and the earliest specific diagnostic indication, the characteristic roentgenographic appearance. On the other hand, a diagnosis of this disease should not be attempted without correlating all the possible facts such as the occupational history, symptoms, signs, laboratory, and roentgen observations.

Of all these methods at reading a diagnosis of silicosis, roentgenograms are the most important. Smith states that where economy of time and expense is desirable it would seem entirely legitimate to limit the examination to a good roentgenogram combined with an occupational history. She goes further to state that

in studies made of silicosis among miners of the Tri-State District of Oklahoma, Kansas, and Missouri there was very little difference in lung expansion between the negative group and the group having third stage silicosis. Moreover, it was found that in 25% of the men with well developed silicosis the lungs were apparently normal by physical examination. Watkins-Pitchford discovered that of a group of 541 miners who had been receiving compensation for silicosis on the basis of a physical examination alone, 41% were found not to have it when x-rays were introduced.

It is interesting that, among granite cutters, four individuals with advanced silicosis had vital capacities for instance more than 92% of normal calculated on the basis of height. The great importance of the roentgenogram in making a diagnosis of silicosis is stressed by most workers in the field. Among these are Cooke, Russel, Panwert, etc.

It may be emphatically concluded that except in the advanced stages it would be frequently impossible to make a diagnosis of this condition without x-rays and for accurate study the two most important materials are in the first place a careful occupational history and in the second place good chest x-rays.

#### *Differential Diagnosis*

In a discussion of the differential diagnosis of pneumoconiosis from other diseases Pancoast and Pendergrass divide pneumoconiosis into three stages from a roentgenological standpoint:

1. In the perivascular-peribronchial-lymph node stage, the hilar shadows are more prominent than usual and of greater diversity and homogeneity. The trunk shadows and linear markings show increased prominence. The appearance is not pathognomonic of pneumoconiosis because it is produced by many other conditions. Furthermore, these appearances should not be ascribed to pneumoconiosis unless shown to have arisen during serial examinations and unless all conditions producing like abnormalities can be excluded.

The following conditions may closely simulate the appearances of this phase of the condition:

- a. Passive congestion of the lungs as a result of cardiac decompensation. In this condition the enlargement of the heart shadow, the abnormalities of the cardiac silhouette especially in the case of mi-

tral stenosis, and the clinical picture serve to establish the presence or absence of passive congestion. It is to be noted that emphysema and passive congestion often occur in the course of all the stages of pneumoconiosis and serve to greatly accentuate the roentgenological appearance of the condition.

- b. Passive congestion associated with coronary thrombosis leads to prominent hilar and trunk shadows lasting from three to six weeks after the attack. This appearance and the clinical evidence of decompensation will clear up when compensation is established. Pancoast and Pendergrass saw one case of this kind mistaken for pneumoconiosis.
- c. Advanced Bilateral Bronchiectasis in which there are dilated bronchii retained secretions and associated tracheo-bronchitis, the appearance is that of greatly accentuated trunk shadows which may simulate this stage of pneumoconiosis.
- d. Asthma may produce various alterations from the normal roentgenographic appearances of the lungs. There may be an intensification of the hilar and trunk shadows and very often an associated coarse mottling of the lung fields. The two conditions may be associated and what might be a very moderate degree of incapacitating pneumoconiosis would have its appearance greatly accentuated by the asthma. It is usually easy to differentiate these two conditions both from a clinical and a roentgenological standpoint.
- e. Malignant metastases in the lungs not uncommonly take on the appearance of greatly accentuated hilar and trunk shadows which closely simulate the appearance of pneumoconiosis.
- f. Polycythemia usually has associated with it an increased prominence of the hilar and trunk shadows due to the engorged pulmonary vessels. On the other hand, high red cell counts, even sufficient to suggest polycythemia are very frequently associated with pneumoconiosis. There seems to be reason to believe that lung fibrosis and pulmonary arteriosclerosis can produce a condition akin to polycythemia and presumably due to impaired interchange of oxygen and carbon dioxide. Yates and Constam state that secondary pulmonary arteriosclerosis involves the larger vessels and may be due to mitral stenosis, emphysema, congenital heart disease and conditions in which the lung volume is reduced as tuberculosis and tumor.



- g. Mycotic infections of the lungs generally produce appearances simulating the nodular phase of pneumoconiosis but occasionally they may be confused with the perivascular - peribronchial - lymph node aspect of the condition. The differentiation may be most difficult or impossible. It is often on clinical grounds that the differential diagnosis must be made.
2. The early interstitial stage of pneumoconiosis is characterized by a rather faint homogeneous haze, appearing first in the right mid or lower lung field and then in the left and gradually spreading in extent. Small discrete dense nodules may or may not coexist. The hilar and trunk shadows are noticeably accentuated. There is more or less interference with diaphragmatic excursion. Aside from the differentiation of a tuberculous pneumonic process in the rapidly developing cases, the only other conditions to cause confusion in the proper interpretation of the homogeneous haze would be an interstitial pneumonitis and a rheumatic pulmonitis. The differentiation is an important one.
3. The nodular phase is productive of the most characteristic roentgenographic appearances of pneumoconiosis. While it is quite typical of the condition, it does not occur in connection with all industries, especially when the intake of silica is unusually copious and the condition of silicosis is rapidly progressive. The characteristic appearance of the slowly progressing type is that of small, dense, discrete nodules throughout both lungs. There is no definite pleural involvement. In the more progressive type the smaller nodules become larger and conglomerate and there are further evidences of interstitial fibrosis obscured by the associated emphysema. In the more progressive type of smaller nodules become larger and conglomerate and there are further evidences of fibrosis indicated particularly by restricted diaphragmatic excursion and often peaking of the domes. The appearance may be simulated by a number of conditions, including tuberculosis of the perinodular type, tuberculous bronchopneumonia and military tuberculosis. The appearance is also simulated by:
  - a. Nodular metastatic malignant conditions of the lungs. Most cases of this kind are readily differentiated from nodular silicosis but occasionally one encounters a case in which appearances are very similar because the metastatic nodules are small, unusually numerous and widely and symmetrically scattered.
  - b. Actinomycosis may occasionally present an appearance like that of nodular silicosis, although the nodules are usually larger and less numerous in comparison with it.
  - c. Spirochetosis may give an identical roentgenological appearance to this stage of silicosis. These various conditions make it apparent that one cannot rely on the x-ray alone for a diagnosis of silicosis, but that the history and the laboratory must be enlisted in making an accurate diagnosis.
  - d. Leptothrix infection may also be difficult to diagnose from a roentgenological standpoint. There are other fungi such as monilia that from an x-ray standpoint cannot be differentiated from silicosis, though in all these conditions a careful search of the sputum, the history, and the course of the disease eventually point to the correct diagnosis.
4. The advanced diffuse or terminal fibrosis stage of silicosis must be differentiated mainly from chronic diffuse tuberculosis or an accompanying tuberculosis is to be determined. Occasionally a case showing massive fibrotic areas mesially located may present an appearance resembling a mediastinal tumor.

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To memorialize the medical profession's "skill and courage and devotion beyond the call of duty" is the purpose of the new prize-contest recently announced by the American Physicians Art Association.

The contest is open to all physicians, both civilian and military, who are members of A. P. A. A. The prizes are sufficiently important to attract some very fine art in all of the principal media, including oil, water color, sculpture, and photography.

For full details, write to the Association's Secretary, Dr. F. H. Redewill, Flood Bldg., San Francisco, Cal.



## STANDARDS FOR HOSPITAL RADIOLOGICAL SERVICES

DR. MAITLAND S. DIRKS

Phoenix, Arizona

*Mr. President, Members, and Guests of the Arizona Hospital Association:*

I appreciate the honor which Dr. Sechrist has given me of permitting me to speak before you on the subject of "Standards for Hospital Radiological Services." I am sorry that Dr. Watkins was unable to bring you his views regarding radiological practice in our Arizona Hospitals because, his experience is both wider and older than mine.

Definitions are tiresome but the use of the term "Radiologist" may not be familiar to all of you. Perhaps, if I am fortunate, when I have finished with what I have to say, you may have a better idea of the meaning of this word "Radiology" than an aunt of mine had when she was trying to console my mother upon my leaving home to study the medical aspects of x-ray diagnosis and treatment in the University of Iowa Hospitals. I had not then been far from home for any length of time. It was not too clear to friends and members of my family what the nature of my work would be. Finally, my dear aunt had this to say to my mother, "It must be terribly hard for you to see him spend all these years studying medicine and then give it all up to fix radios." Radiology is that branch of medicine which deals with the use of x-rays and the problems of radium. Because of limitations of size and equipment of some hospitals, many physicians qualified in Radiology are forced to limit their work in the hospital to only a portion of what they are potentially trained to do.

Dr. Boyd has already addressed you on the subject of "The Doctor's Relation to the Hospital." Therefore, I will direct my remarks more toward the operation of the hospital Radiological department. Proper standards vary with the size, location, and staff of each hospital, but detailed suggestions and principles for adequate service have been set forth from time to time by Council on Medical Education and Hospitals of the American Medical Association. This was recently reported in the Journal of The American Medical Association for

1939. The American College of Radiology and the American College of Surgeons have assisted in preparation of detailed standards. Assistance in coordinating this work has come from the report of the Council on Community Relations and Administrative Practice of the American Hospital Association, published in "Hospitals" for the year 1937. I refer you to these reports rather than attempt a detailed analysis of necessities for each class and size of hospital.

General principles apply, however. The responsibility for all radiologic examinations must rest on the physician-radiologist, who is head of the department. His findings and conclusions for all examinations should be placed in the patient's chart. In the case of the larger hospital in the larger centers, this director should be a physician who is qualified by the American Board of Radiology in his specialty. If the hospital is 200 beds or larger, his full time services are required. If the hospital is smaller but unusually or steadily active, it is desirable to have a full time qualified Radiologist or Roentgenologist even in cases where the bed capacity may be as low as 125 to 140 beds.

Smaller hospitals need a well organized x-ray department but proportionately less of a trained radiologist's time. He should give the hospitals a definite schedule of hours when he will be in the department for consultation. This should coincide with the day and time of the staff meeting where feasible.

Where no trained director is available, it is desirable for a member of the hospital staff to take one of the short intensive courses in x-ray diagnosis only, at such a center as Columbia University. Where this is not possible, the physician-director should have adequate equipment for good fracture work and the simplest diagnostic procedures. It is probably best that he not attempt often to do the more intricate procedures involving contrast media. I do not need to labor the point that x-ray treatment is dangerous to the patient and personnel in inexperienced hands. A physician not prop-

erly qualified should not be urged by his staff to undertake this phase of radiology.

**PERSONNEL:** In hospitals of 35 to 50 beds, taking general cases, the department will need the assistance of an x-ray technician whose duties are devoted for at least part of the day to obtaining films of proper quality for correct interpretation. Where only one person is needed, a technician who is a nurse makes operation of the x-ray department smoother and more satisfactory to patient and doctor. Larger departments will need one or more technicians full time plus the assistance of a secretary and often a nurse. The latter is most needed where therapy cases are taken for treatment.

**FUNCTIONS:** The first function of a hospital Radiological department is to provide the means by which a radiologist or director may render adequate service to the hospital patient. This should mean in smaller hospitals a unit of sufficient power to produce a chest film in 1/10th of a second's time with the patient 6 feet from the x-ray tube. There are many other desirable features including the ability of the apparatus to provide for fluoroscopy of the patient in the upright and in the horizontal postures, as well as for what we term "grid" films of good quality in the same positions.

The Radiological departments of hospitals of 100 to 125 bed capacity and larger, should in general, include facilities for obtaining excellent diagnostic films which smaller machines do not produce, and also provide the three usual types of x-ray therapy. These are superficial, intermediate, and deep therapy. Where x-ray therapy is used it is often best to include a minimum of 50 milligrams of radium element in containers chosen by the radiologist. This may sound at first as if three classes of x-ray therapy units would be required. Fortunately, this is not so. A unit primarily intended for adequate diagnostic work or radiographic work as it is technically called, can also be used, when properly designed, for superficial x-ray treatments as well. Dr. Salisbury has such a unit at Ganado and we have the same type of machine at St. Joseph's Hospital. This type of machine, of which there are several makes, sells for around \$4,500 and is capable of making all ordinary x-ray examinations with good resulting quality. The super-

ficial x-ray therapy feature is most useful when properly supervised.

Deep therapy apparatus is primarily intended for operation at around 200,000 volts which we term 200 kv. but, it is also possible on most present day deep therapy units to adjust the mechanism for use in cases requiring intermediate therapy around 120 to 140 kv. This description is admittedly technical but I give it to show that a hospital can save money by first obtaining the advice of a radiologist in planning a department, and then approving for purchase a multi-purpose equipment which costs no more initially. A deep therapy machine does represent an investment of at least \$5,500. But, since it is applicable only to hospitals which require the services of a trained radiologist for a minimum of two to three hours daily, the volume of business usually can be counted on to justify it. I am aware that space limitation has prevented several of our larger Arizona Hospitals from including adequate x-ray therapy service. I hope soon to see this situation corrected when construction can again be undertaken. X-ray therapy should be looked upon as a distinct need of the patient and provided where possible. In my opinion it should be considered essential to adequate hospital care where a qualified radiologist is department director and the hospital records show a sufficient number of the type of cases admitted which are amenable to treatment. Even when the revenue simply offsets base cost of operation of the equipment and sufficient additional perhaps to repay or nearly repay depreciation. This may seem to you folks in hospital management as unsound, but I would point out that Groover has shown in his widely read report on cost of operation of Radiological departments that: first, in those cases where 50% of the professional time and effort and 50% of the departmental operation cost go to operation of the therapy section, yet, only 30% of the revenue is obtained from therapy cases. But, in such balanced departments the entire department is financially sound since total operation costs can be kept between 33 and 35 per cent of the gross income, since 70% is derived from diagnosis.

I will ask your tolerance to mention two more figures which must be important to you

when considering the operation of your hospitals. On the average it is agreed that in the smaller hospitals of less than 75 beds, depreciation of x-ray equipment amounts to about 15%. For larger hospitals this figure seems to be smaller and around 10% for installations which place two-thirds of their emphasis on diagnostic work and one-third on therapy. Of course, an unbalanced percentage of therapy work above 50% would increase this figure again.

**RECORDS:** In addition to complete records of the opinion of the radiologist kept in the patient's chart and in the department for reference, a word should be said about care of films.

These should be filed systematically by number or name, and crossed-indexed if possible along with reports. Physicians should be free to borrow them for reference, but they should return them promptly. You people can help your hospitals and the radiologist by helping to impress the patient with the fact that he bought medical opinion and hospital service and did not purchase the film any more than any other part of his permanent hospital record. Seven years is now considered long enough to keep x-ray films. The records should be kept indefinitely.

It has been a great privilege to speak before you, and I thank you.

## THE CLINICAL VIEW OF SILICOSIS

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**I**N a consideration of the clinical development of silicosis it is noteworthy first of all that a good deal of time elapses ordinarily between the beginning of exposure to silica dust and the appearance of the first evidences of silicosis. Reports vary, but the average amount of time required for the development of the first symptoms may be roughly estimated to be from five to ten years from the beginning of exposure. The duration of the disease from the beginning of symptoms until the end may also be roughly averaged at about ten years—six to eight years with moderate symptoms, moderate impairment of working power, and little apparent change, and then a year or so of complete disability and prostration.

The duration of the disease as well as the time that it takes to get started vary inversely largely with the intensity of the silica bombardment to which the worker is subjected. The heavier the load of silica in the inspired air the earlier the symptoms will begin and the shorter will be the course of the disease. Miners, for instance, using air drills in hard rock are affected more quickly than those working without air drills in softer rock that contains less silica. Similarly a form of "acute silicosis" is recognized that occurs chiefly where scouring soaps and powders are made by mixing finely powdered silica with alkaline soap. It was at first thought that the chemically ad-

mixed alkali made the silica more deadly, but it is now believed that the rapidly fatal acute silicosis is caused simply by the massive doses of silica inhaled in the very dusty process—doses heavier than those encountered in other industries. Series of cases of "acute silicosis" are reported that were exposed for from eight to twenty-one months, that developed symptoms in from nine to twenty-four months after the beginning of exposure, and that terminated fatally in from fourteen months to seven years.

Once started the silicotic process seems to be able to go ahead under its own steam. Although, of course, exposure may be continuous until symptoms appear and until the power to work ceases, this is by no means invariably or necessarily so. It is very important to recognize that exposure to silica dust may have ceased finally before any symptoms of silicosis appear, and that having thus appeared the disease may progress to a fatal termination without re-exposure. One case is known to have been exposed for four months, to have had an interval of twenty-three years free from exposure, and then to have developed silicosis; and another, to have been exposed for four years, with a free interval of fourteen years following, before the appearance of the disease. Other cases with latent periods up to fifteen years are reported.

The composite clinical picture of simple, uncomplicated silicosis has been arbitrarily divided into three stages. Throughout the entire course however, the cardinal, predominating symptom is *dyspnoea* upon exertion.

The first stage begins very insidiously. The first symptom to appear is almost invariably slight *dyspnoea* upon exertion, generally more noticeable at the beginning of the day. There is perfect comfort apart from exertion—general health, nutrition, muscular development, hemoglobin and red cells, all are excellent. There is no disability. There may or may not be a bronchitic non-productive cough, and rarely this is the first symptom reported. The first physical sign noted is very slightly decreased expansion of the chest, bilaterally, and fluoroscopically the first change is a very slight decrease in the amplitude of the excursion of the diaphragm, bilaterally (thought to be due to inelasticity of the lung as much as to pleural thickening or adhesion). Later slight dullness on percussion may be detected over the roots of the lungs. Inspiration may be heard to be thinner, higher pitched, and harsher than normal, and expiration somewhat prolonged but fainter than inspiration. There may be a few basal rales. There may rarely be asthmatic wheezing.

In the second stage there are definite *dyspnoea* on exertion, pains in the chest, sometimes expectoration, sometimes a dry cough that may lead to vomiting. There is definite impairment of working capacity. All physical signs previously noted are more pronounced. Chest expansion, if measured, is found to be less than before.

In the third stage, *dyspnoea* is marked and distressing upon the slightest exertion. Cough, sometimes with copious sputum and occasional hemoptysis is frequent and annoying. Rarely there is fever. Evidence of the *cor pulmonale* appears in accentuated pulmonic second sound, cyanosis, and in right axis deviation in the E. K. G., and later in rapid pulse, cardiac dilatation, and decompensation. Possibly a third of the cases have curved nails and clubbed fingers. There is loss of weight and strength, and serious and permanent impairment of working capacity. Inspection shows a chest flattened and smoothed and frequently not barrel shaped, with a great decrease in expansion bilaterally

and at times retractions substernally and at the bases laterally. Rarely the signs of cavity formation may be detected. The picture is of the rigid lung of fibrosis mixed with emphysema.

Asbestosis is a somewhat distinct form of silicosis that presents some minor differences from it. Observers report that the skin may take on a leaden, pasty hue, and the early appearance is less robust. Cyanosis and slight clubbing of the fingers appear early and there is a greater tendency to right heart insufficiency. Hemoptysis is more frequent. Many other symptoms appear to be milder. There is less complicating tuberculosis. The sputum may contain "asbestos bodies", and asbestos fibres frequently become imbedded in the skin and cause "asbestos corns."

Spontaneous pneumothorax occurs in silicosis, sometimes bilaterally. It is not frequent but must be considered a complication of the disease. It is thought to be due to the tearing of an emphysematous bleb by a pleural adhesion or to the advance of ischaemic necrosis the result of dust infiltration. It occurs oftenest in the apical region.

Bronchogenic carcinoma is reported in connection with silicosis, allegedly caused by irritation from silica.

The prognosis in simple silicosis is bad. It is commonly said that "no improvement can be expected in silicosis." Most cases go on to a fatal termination. However, studies have been made of cases in sanatoria and a percentage has been reported improved while under treatment. Some workers incline to the belief that if the rest treatment given to tuberculosis were given to silicosis long and faithfully enough, the prognosis might be more hopeful.

Silica is of very great importance and interest for two reasons. First, of all the organic and inorganic dusts encountered in industry silica alone (with its magnesium silicate, asbestos) produces a distinct, progressive disease entity, which is now recognized as a deadly industrial hazard. Although some students hesitate to admit the existence of a simple silicosis uncomplicated by pulmonary infection, it is pretty generally accepted that such cases do exist and have been exhaustively studied both clinically and post-mortem without the discov-



ery of any evidence of tuberculous or other infection. In the second place, however, silica alone of all the organic and inorganic dusts does stir up a most extraordinary susceptibility to tuberculosis. The fact is that sooner or later most cases of silicosis do develop pulmonary tuberculosis, either because silica reawakens previously existent quiescent foci or heightens susceptibility to bacilli subsequently implanted. It has long been a dictum that seventy-five per cent of silicotics die of tuberculosis. This percentage is now thought by some to be a little high, and it is considered that the cor pulmonale leading to congestive heart failure cuts it down somewhat, but all agree that a very substantial proportion of silicotics do die of pulmonary tuberculosis.

When silicosis is complicated by tuberculosis the situation becomes graver both for the patient and for his contacts. For a while tuberculosis may coexist with silicosis without changing the picture and without detection except with great difficulty, but soon there is a change and the course goes rapidly downward. Toxic

symptoms such as fever and rapid loss of strength and weight arise, sputum may become positive, cavities may form, frank hemoptyses are not infrequent, pleural effusion may develop, tuberculous lesions may appear elsewhere in the body, and a study of serial x-rays over a period of time may show the developing tuberculosis. The physical signs differ somewhat from those of simple tuberculosis and suggest a bilateral, symmetrical fibrosis over-shadowed by emphysema, with consolidation and excavation in the hilar and basal regions predominantly, of both lungs.

It is hoped that nothing in this resume will encourage the belief that silicosis, with or without tuberculosis, is an easily diagnosed affair. Actually, every case confronts the physician with a diagnostic problem that is frequently difficult in spite of all the help that a careful history and a competent roentgenologist can give, and that is always of urgent importance not only to the patient but also to the public health.

## APPENDICITIS COMPLICATED BY RUPTURE OF THE INFERIOR EPIGASTRIC ARTERY

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**S**PONTANEOUS hemorrhage from the inferior epigastric artery is not rare. Its occurrence on the right side simulating acute appendicitis is interesting and adds one more factor to the diagnosis of the latter condition. In that a search of the records fails to show that the two entities have been reported as occurring simultaneously, this situation is unusual.

Rupture of the inferior epigastric artery may be due to trauma or may occur spontaneously. It has been reported as a post-operative occurrence but in these cases may be considered as probably the result of trauma to the belly of the rectus abdominis. Cases following a severe coughing attack are considered traumatic, if immediate.

Mailer, writing in the British Medical Journal in 1936, mentions the possibility of hemorrhage from the right inferior artery being erroneously diagnosed as acute appendicitis. This is especially possible where the rupture is spon-

taneous, although Hilgenreiner in 1924, referring to a group of seven cases following violent exercise among recruits refers to the possible mistake in diagnosis that may occur.

The following case is reported as of interest as it was associated with definite inflammation of the appendix.

The author was called April 30th at 9 A.M. requesting an appointment. The patient stated that he thought he was having an attack of appendicitis. He was seen at 10 A.M. He was a robust male, age 56, who seemed in great pain. He gave a history of being awakened at 3 A.M. with a sharp pain in the right lower abdomen. He was nauseated and vomited shortly afterwards. The pain was severe enough to cause him to draw his right leg up for ease. Because he had had previous attacks he decided not to call the doctor until morning. He stated that he had had a severe cold for the past ten days, accompanied by a paroxysmal cough. He



had retired at 10 P.M. and had had several coughing spells shortly after retiring.

His previous history was negative except for several attacks of lower right abdominal pain, diagnosed as appendicitis by the attending physician. The last about 15 months ago. He had remained in bed with each previous attack for several days, without food, an icebag on his abdomen. For the past year he had been very well, had had no pain in the abdomen, no epigastric distress, and his bowels had been fairly regular. He was employed as a telegraph operator, worked long hours and was irregular in his eating habits. He had partaken of no unusual food the day preceding the attack.

Examination showed a florid heavy-set man lying quietly with his right leg drawn up. His expression was anxious. The temperature was 99.2, pulse 94, respirations 20. The blood pressure was 154/90. There was no noticeable sclerosis of the radials. The heart was not enlarged to percussion, there was no murmur or other evident abnormality. The expansion of the chest was full and equal on both sides. The breath sounds were somewhat harsh and there were scattered S&S rales throughout both lung fields, especially at the bases. Fluoroscopy of the chest and heart was negative. The remainder of the examination, except the abdomen was negative. He gave no history of kidney, bladder or prostatic disease. The urine showed Albumen 2 plus, pus cells 1 plus, and a few granular casts. He had had a negative Wassermann about six months before.

The abdomen was full, there were no scars, and no evidence of hernia. Over McBurney's point there was increased fullness that extended medially into the lower portion of the right rectus. On palpation there was definite tenderness over McBurney's point that extended medially over the same area. A very definite muscle spasm was noted over the same area. The tactile test was clear cut and positive below a line drawn parallel to the umbilicus. Percussion showed increased tympany in this region, and very light percussion over McBurney's point and medially elicited quite marked pain and spasm, and provoked nausea.

He was sent to the hospital with a diagnosis of acute appendicitis and blood counts were ordered. He was given no food, and ice bag was

kept on his abdomen and he was given codeine by hypo for cough. The first count showed, red cells 4,950,000, total white 9,750, polys 65. About six hours later the second count showed, red cells 4,870,000, total white 11,750, polys 71%. At this time his abdomen was re-checked. The tenderness was possibly more marked. The swelling before described was more noticeable, the muscle spasm was increased although he stated he had much less pain. Because of the increased swelling and low white count it was decided to watch him for 24 hours. He was given water freely and did not vomit further, though he had some nausea. He was given codeine enough to control the cough. The following morning the blood count showed, red cells 4,700,000, total white 12,400, polys 74. A further check of the abdomen showed an increase in the area of swelling with possibly some increase in the tenderness. He had a tendency to frequency but was otherwise comfortable. His temperature was 99, pulse 84, resp. 18. The second urinalysis was essentially the same. An enema gave a return of a soft copious stool and afforded him some relief. He rested quietly all day and was prepared for surgery the following morning.

Although we would not ignore the possibility of an appendix, we felt that with the increasing swelling, great tenderness extending into the lower part of the right rectus belly, and falling red count that we might find a ruptured inferior epigastric artery.

Under spinal anaesthesia he was operated the following morning. A right rectus incision was made. As soon as the fascia was exposed it was observed to be bluish in color and very tense. On careful incising it was found to be very friable, and as it separated a large blood clot exuded from the sheath. The blood clot extended from the umbilicus downward. It surrounded the rectus muscle and extended as a large mass into the pre-peritoneal tissues. The rectus muscle was fragmented badly. When the clot was removed the deep epigastric at a point one half inch within the sheath was found to be spurting briskly. The accompanying vein was torn. Both were doubly ligated. The peritoneum was opened and the blood clot was observed to extend under the peritoneal reflection on the bladder. The caecum was lifted

ed. There was a moderate amount of cloudy fluid in the fossa, the appendix was retrocaecal, injected and swollen. A fairly heavy band of adhesions was found midway. The appendix was removed and stump inverted. The abdomen closed in layers without drainage.

Convalescence was uneventful. He was given sulfamerazine to clear up the bronchitis and discharged on the 9th post-operative day. On the third post-operative day the urine was entirely clear.

This case was interesting as a study in diagnosis. Operation was deliberately postponed in order to observe events, though we were prepared to intervene at any time if events indicated. The classic picture as presented when the rectus sheath was entered could not fail to excite comment, with tremendous fragmentation of the rectus muscle. The infiltration of the hemorrhage into the peritoneal reflection above the bladder certainly explained the frequency. It was not so easy to explain the urinary findings solely on the basis of the hemorrhage. It was felt that the paroxysmal cough was probably indirectly the cause of the rupture, although the delay in the onset of the pain was not so obvious, as it had been sudden and acute and followed any recalled coughing spell by quite an interval. No previous report of rupture of the right inferior epigastric artery associated with acute appendicitis has been noted in the literature.

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## Cancer Section

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### CANCER OF THE PROSTATE

By John W. Pennington, M. D.

In 1941 one of the outstanding contributions in the treatment of cancer was presented to the American Urological Association by Dr. Huggins<sup>1</sup> of Chicago. This treatment of prostatic cancer was the result of his application of castration in an effort to change the serum acid phosphatase content in the blood of patients with enlarged prostates. At the same meeting Munger<sup>2</sup> of Lincoln, Nebraska, presented a paper advocating the x-ray castration of patients with carcinoma of the prostate. Later the natural outcome of these methods was the use of

estrogenic substance in the treatment of prostatic malignancy.

The etiology of prostatic cancer is no more definite than any other of the ordinary malignancies. Wilhelmi<sup>3</sup> of St. Louis feels that the serum acid phosphatase is of no value in early diagnosis of prostatic cancer and a more standardized ratio must be recorded before the test is of definite value to the surgeons. He feels that urinary acid phosphatase from Demuth's work appears to be more accurate and constant than the serum acid phosphatase in evaluating the stage of the disease.

T. J. Sullivan, E. B. Gutman, and Alexander B. Gutman<sup>4</sup> attempted to correlate the serum acid phosphatase determination as a guide in the diagnosis of prostatic lesions. To date there have been few reports in regard to the type of prostatic malignancies that can be expected to show good results from the various methods of treatment. Wilhelmi feels the undifferentiated type of carcinoma and those cases with light weight testes have a poor prognosis. It is hoped that a standardization may be worked out as a guide to the type of therapy indicated and the results expected.

In the treatment of prostatic malignancy one must refer to the pioneer work of Dr. Hugh Young with his radical perineal prostatectomy, which has so ably been carried on by Hinman, Belt, and others. In a very early malignancy with no extension, or little extension, the radical perineal prostatectomy offers a very good chance of cure, but this operation should only be done by surgeons who are capable perineal anatomists. The use of transurethral resection in the treatment of malignancy of the prostate is well discussed by G. J. Thompson<sup>5</sup> of Rochester, Minnesota. He concluded that in a high percentage of cases complete freedom in the voiding of urine is provided by transurethral resection and that this freedom exists until the patient dies of metastasis or other unrelated disease. Thompson felt that if transurethral resection is combined with Huggins procedure of castration, the results in the future should be superior to those in the past.

**CASTRATION.** This procedure alone is applicable in those patients who have an absence of marked bladder distress and disfunction, although it probably will afford a great deal of

relief, if not entire relief, even though there is considerable residual urine.

Estrogenic substances can be used orally, by pellet implant, or subcutaneously by hypodermic injections; and, if adequate dosage is given, offers considerable relief, possibly even arresting the cancer.

An evaluation of the results obtained from the various forms of treatment was very satisfactorily given by Nesbit and Cummings<sup>6</sup> of Ann Arbor, Michigan, in the January 8, 1944 issue of the Journal of American Medical Association. In a follow-up on 75 cases of prostatic carcinoma treated by orchiectomy they found there was a very spectacular early relief of pain usually obtained in from 20 to 72 hours, although occasionally the pain subsided over a period of several weeks. Many patients gained as much as 50 to 60 pounds in weight. They found urinary obstruction, even acute retention, responded satisfactorily in all cases. In their series of 75 patients 10 derived no benefit at all from orchiectomy. 45% of the cases obtaining relief remained free of symptoms up to 36 months after the orchiectomy, but 21 patients who had immediate satisfactory results had recurrent symptoms of advanced disease. Nesbit and Cummings felt that it was logical to conclude that the maximum benefit to the patient may be derived by delaying endocrine treatment until indicated by the onset of symptoms arising from advanced or metastatic lesions. Only in this manner could a longer period of palliative relief be assured.

In the writer's opinion, orchiectomy should be done as soon as the diagnosis is established. I feel the only way to make a positive diagnosis is by microscopic section of the tissue, which is usually best obtained by a transurethral resection. If bony metastasis are demonstrated, the use of estrogenic substance should be started immediately after orchiectomy and continued indefinitely. If metastasis recur or become painful, deep x-ray therapy often will alleviate the pain from these lesions. In my own experience x-ray castration by exposing the testicles has not proven as satisfactory as the other methods. It is my opinion that between 50% and 60% of patients with carcinoma of the prostate can be given quite satisfactory relief by using one of the above mentioned forms of

therapy or by a combination of two or more of them.

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### U. S. SOLDIERS FITTEST EVER, GENERAL SAYS

#### *Army Surgeon General Reports Falling Death Rate From Disease and Wounds*

Today's American soldier is the healthiest to ever take the field against the nation's enemies, Maj. Gen. Norman T. Kirk, Army Surgeon General, maintains.

The rigid screening process by which both civilian and military doctors have weeded out the physically and emotionally unfit is largely responsible, he said in a nationwide broadcast of "The Doctor Fights," Tuesday night (June 20) CBS program, sponsored by Schenley Laboratories, Inc.

In previous wars, the general reported, pneumonia has been responsible for many deaths but "during the winter of 1943 something under 1 per cent of cases of pneumonia in the United States armed forces died of that disease."

"Meningitis, which 50 years ago had a death rate approximately 80 per cent and which 25 years ago had been lowered to a death rate of 40 per cent, has today in our armed forces a death rate approximately 5 per cent," he continued. "In one great military installation there have been reported 76 consecutive cases of meningitis without a single death."

These figures, Gen. Kirk added, can be applied to most of the common diseases that affect mankind, particularly under military conditions.

"Never before has an army more physically fit than this one taken the field against our nation's enemies," he declared.

The low death rate, he continued, was a tribute not only to physicians but "particularly also the magnificent organization for medical research which is carried on during the war period."

"This research," Gen. Kirk said, "has been reflected in the exceedingly low death rates from wounds of all men not killed outright in the use of such remarkable products as blood plasma, the sulfonamide drugs, penicillin and in the preventive measures for the control of measles, meningitis, typhus and many tropical diseases."

# ARIZONA MEDICINE

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## Editorials

### Plans for Medical Care

In the nation-wide poll recently completed by the National Physician's Committee, 63% of all the people voiced the opinion that methods could be devised for easier payments of medical care costs. This is not to be interpreted to mean that the American people want their medical care cheapened. A doctor receives no credit for trying to save his patient any money. Probably nine out of ten patients want complete examinations, including x-rays, blood tests, and all other available laboratory tests. And, any attempt on the part of the physician to eliminate what he might consider as unnecessary expense is very apt to result in a dissatisfied patient. To a great majority of the American people a trip to the hospital and a major operation is an event of a lifetime. It corresponds to a wedding, or a funeral, or some anniversary. They want a private room, three special nurses, and wish no chances taken. Frequently a surgeon sees his fee going into these luxuries, and then has to wait until the family pocket-book is replenished before he is paid.

Those among us possessed with wisdom and vision assure us that Socialized Medicine is coming. When the Government pays the bills as is outlined in the Wagner-Murray-Dingall Gill, our imagination is captured when we visualize some political appointee in the front office of a hospital deciding who shall have a private room and who shall be assigned to a

ward bed, and how long certain patients shall retain their three special nurses.

To meet the demand for easier payments of medical care, the entire American Medical Association have given their approval of voluntary prepayment, non-profit insurance plans. At the present time there are some 200 such plans in operation, with a total membership of 25 million. The National Physicians Committee have signified their intention to promote these plans until a membership of 100 million is reached. These plans are formed to fit the particular situations in a specified community. e. g. a plan organized for an industrial center would not operate very satisfactorily in a farming community. The provisions of the different plans vary also, depending on the membership of the unit, and any reserves they have set up. None of them can be said to be ideal, but it has been pointed out that it required 40 years to perfect Old Line Life Insurance. But one of the main pre-requisites of any plan must be that it be a non-profit organization. In this way a certain percentage is set up for operational expense, and all the remainder goes for benefits to the members.

Of these 200 plans, some 70 belong to the Blue Cross, with a membership of about 15 million. The Blue Cross have set up certain standards and requirements, which add much prestige to its membership, and practically make it a nation-wide Organization.

The Associated Hospital Service of Arizona was organized about 5 years ago. With the advent of the war it became dormant. But the Board of Directors are working to make it active again. This should be one of the foremost objectives in post war medical planning for Arizona. It is working to meet all the requirements for admission to the Blue Cross and we hope soon to be able to announce acceptance by this Organization.

### Good-by, Old-Timers!

The American Pharmaceutical Association announces a complete revision of "The National Formulary"; new edition expected to be ready late in 1945.

Marked changes in the context will be noted, since it is stated that nearly one-third of the drugs mentioned in the older editions will be



deleted, and that some one hundred fifteen new names will be added. One radical change will be the substitution of English names for those formerly appearing in the Latin language. The latter, however, will in many cases follow the English term, as a secondary title. Metric dosage will be emphasized, although the present apothecary dosage will be maintained, it being felt that many physicians were not ready to drop the use of the old dosage formulae. (This might be considered as a friendly gesture to the oldsters.)

It is planned to standardize the enteric coated medications, since the claim often has been made that many of these preparations are misnamed. Also, it is said that the new Formulary will be more "self-contained," making it unnecessary to look up many items in the Pharmacopoeia.

The various state pharmaceutical associations are being asked to go over the proposed list of deletions and suggest their own ideas about them.

Looking over this list we find many old-time friends, several of which we have been wont to include in our armamentarium. A number of these drugs would be classed as "strangers" by the more recent graduates in medicine, since they are not even mentioned in most modern courses of therapeutics. However, to the oldster they have been stand-bys for years, and they will miss them if they have occasion to consult the new edition of the Formulary.

For several years past the Council on Pharmacy and Chemistry of the American Medical Association has issued a volume entitled "Useful Drugs." With each succeeding volume more and more drugs are dropped from the list. Now comes the Pharmacist's Bible, with approximately one-third of the formerly-mentioned drugs dropped from the list.

Among the old-timers found in the medicine kit or dispensing room of the older members of the profession, but are now termed passe, might be mentioned Blue Flag; Bryonia; Calendula; Chionanthus; Echinacea—there will be lot of mourning over the passing of this one—Elix, Buchu and Potass. Acetate; Elix. Pepsin and Bismuth; Phytolacca; Plaster of Cantharides; Pulsatilla; Tr. Capsicum and Myrrh; Tr. Cimicifuga and Tr. Sanguinaria. There are also many others dear to the heart of the old-time

doctor, many of which we swore by and declared that he could not practice without.

All of this means that chemotherapy is coming into its own; or does it mean that we have a chemotherapy wave—one that will ride the crest for some years and then give way to something new? We doubt it, since chemotherapy offers so much and the field is yet almost virgin.

As one of those who have been in practice for more than four decades, however, we pause to pay tribute to a list of drugs that we, at least, thought would be with us at all times. Compelling us to become modern is a jolt, but we can take it!

Reprint from The Journal of the Indiana State Medical Association.

## MEDICO - LEGAL MEDICINE

### IN THE SUPREME COURT OF THE STATE OF ARIZONA

UDALL, Superior Judge:

This is a certiorari proceeding under the Workmen's Compensation Law, Article 9, of Chapter 56, A. C. A. 1939, brought to this court by the Mountain States Telephone and Telegraph Company, a corporation, hereinafter termed the petitioner. It seeks a reversal of an award made August 12, 1943, to Edna S. Mitchell, applicant, for death benefits payable to her for the support of herself and their four minor children then aged 12, 10, 6 and 4 years, respectively. No question is raised as to the amount of the award.

The petitioner-employer was insured with the Industrial Commission of Arizona, as the insurance carrier. Admittedly at the time of the injury both the employee, Mitchell, now deceased, and the employer were subject to the Arizona Workmen's Compensation Law and to the jurisdiction of the Commission. Application for rehearing was denied on September 3, 1943, and within the time allowed by law this proceeding for review was initiated.

There are but two questions raised: (a) Does the evidence in the case support the finding of the Industrial Commission of Arizona that the deceased employee died as a result of carbon tetrachloride poisoning; (b) Did the deceased die as a result of an accident arising out of and in the course of his employment within the true meaning of the Workman's Compensation Law of Arizona?

If this court finds the answer to either or both of these questions is in the negative, then the award should be set aside; on the other hand, if the answer to both questions is in the



affirmative the award should be sustained.

A proper determination of these questions requires a close review and analysis of the evidence as well as the application of the statutory law to the peculiar facts of this case.

While there is some conflict in the medical testimony, we state the evidence in its strongest light in favor of the claim of the applicant. The Commission having made an award to her she is entitled to have the evidence thus considered by us.

Clarence Mitchell, for whose death benefits were awarded by the Commission, was a white American, aged 38 years. He had been in the employe of the petitioner for several years, maintaining telephones.

Prior to his last illness, which occurred late in the month of December, 1942, the general health of the deceased was good.

His employment record during the year 1942, based upon a 40-hour week, was nearly perfect. He was absent from work on account of illness only two days during the year.

On or about October 1, 1942, the deceased was assigned to the work of overhauling and repairing telephone switchboards maintained by the petitioner. As a recondition repair man he had worked at various locations prior to December 24, 1942, when he was assigned by the petitioner to service the switchboard at the plant of the Goodyear Aircraft Corporation, at Litchfield Park, Arizona.

In the work that he was doing subsequent to October 1, 1942, the deceased was required to and did use small quantities of carbon tetrachloride at different times and under varying conditions, without any apparent ill effects until he went to work on the switchboard at the Goodyear plant.

Specifically his work involved cleaning the contacts or terminals in the switchboard relays. The space between the contacts in the relays being only 30/1000 of an inch, the usual method of cleaning same was to dip a wooden tooth pick in a small bottle of the carbon tetrachloride and apply the chemical to the points. Ordinarily the actual use of the fluid was not more than five or ten minutes at any one time and it would be so used at intervals of six or seven times a day. The record does not disclose as to what amount or how frequently the deceased used this fluid on the Goodyear job. This work required close application and scrutiny.

The switchboard upon which the deceased was working at Goodyear was then located in a room  $12\frac{1}{2} \times 11\frac{1}{2} \times 9\frac{1}{2}$  feet, which was walled off from the officers' conference room by plywood. The ventilating system was so installed that it was not possible to ventilate the telephone switchboard room without ventilating the officers' conference room. There were employed in the telephone switchboard room, two

telephone operators, a relief operator, and two girls operating the teletype machine, and there frequently were as many as ten people in the room.

The telephone switchboard room was a dark room with no windows. It had two small lattice vents near the floor; there was a trap in one corner which could be opened for an escape in an emergency. There was one door which opened near the machine shop, but by reason of the noise from the machine shop it was necessary to keep the door closed, in order for the switchboard operators to perform their work. The only ventilation in the room came from a ventilator in the ceiling through which heat from the gas furnaces, or cold air could be brought in the room. This was controlled by a switch. When the cold air was turned on, the officers in the conference room, which was amply ventilated, directed that the switch be thrown and the cold air turned off. The telephone switchboard room was always stuffy and as testified to by some of the women operators "it was almost impossible to sit in there, it was choking and suffocating, and practically all of the employees were ill, suffering from colds all winter." In passing it might be noted that the quarters then in use were temporary. Later the exchange was moved to more commodious and better ventilated quarters.

While performing his work at the Goodyear plant, the deceased worked in small, confined quarters, about  $2\frac{1}{2}$  feet wide, between the switchboard and the cable terminal box. This space was further confined by the wall at one end and the teletype apparatus located within a few feet of where the deceased was working at the other end. The space where he was working was just barely large enough to admit his body and tool box and to permit him to squat down and sit on a box and do his work.

The telephone switchboard room was illuminated by fluorescent lighting but the deceased required an electric light back of the switchboard to see what he was doing. The light, of course, was productive of heat, as were the generator, batteries and motors located in the cable terminal box.

Carbon tetrachloride is highly volatile and heavier than air. Its tendency is to seek the lower levels of the room. Chemically it very closely resembles chloroform.

When the deceased was working with carbon tetrachloride the odor was prevalent throughout the room, to the extent that the switchboard operators complained of feeling nauseated and ill as a result thereof, and called upon him to stop using it. One of them testified "they hated to see him come to work because we knew we were going to get that odor again." When the operators complained of not feeling well, deceased held the bottle across the switchboard and let them smell the contents and told them

that this stuff he was using was what was making them and him ill.

On December 24, after working with carbon tetrachloride under said conditions, deceased went to Dr. Enfield, the plant doctor, complaining of gastro intestinal discomfort and gaseous indigestion. He was given a prescription for indigestion and when he did not improve, he telephoned Dr. Armbruster, the family physician, and requested him to remain after office hours.

The deceased called on Dr. Armbruster after five p. m. December 24th, and advised the doctor that he had been working with carbon tetrachloride. He complained of the same symptoms mentioned to the plant physician, plus a tight feeling in his chest, some cough, pain in his back and abdomen, and chilliness. He also told him that he had become acutely sick on the job. The doctor made no investigation or examination, accepting the symptoms the patient gave, he gave him a prescription and sent him home.

The deceased was sick all day Christmas. It had been his ambition for years to get the particular kind of job he was doing, and he returned to work at the Goodyear plant on December 28, 29 and 30, on which latter dates he frequently came from behind the switchboard holding his sides and complaining of his chest; and, would sit down holding his side and complaining, "I hurt so bad all over my chest—that stuff in the bottle kind of gets me."

On December 31st, on his way to Doctor Armbruster's office, the deceased collapsed and fell several times. He saw the doctor, complained of the same symptoms he had on December 24th, and some additional ones. He was not hospitalized, due to overcrowded hospital conditions. On his way home the deceased again collapsed and had to be assisted. About nine p. m. January 1, 1943, he was admitted to the Good Samaritan Hospital, with further additional symptoms and died at six a. m., January 2, 1943.

In the death certificate, Dr. Armbruster, based upon an incomplete autopsy report, diagnosed the cause of death as "amyloid leukemia"; and subsequently, after the full report was made, the diagnosis was changed to "poisoning and death caused by carbon tetrachloride."

Dr. Maurice Rosenthal, a graduate of a medical college of Virginia in 1926, with subsequent special studies, training, and experience in pathology, and the resident pathologist at the Good Samaritan Hospital, Phoenix, Arizona, examined and performed an autopsy on the body of decedent on January 2, 1943. He reported his findings and conclusions as: 1. Nephrosis, toxic acute, (a swelling of the kidney); 2. Necrosis of liver, central, (a general deterioration); 3. Subepicardial hemorrhage, (hem-

orrhage under the pericardium); 4. Edema of brain and brain stem, (a swelling, an increased fluid condition). (Note: The meaning in plain English of the medical terms by the Doctor appear in parenthesis, supra.) The Doctor confirmed his conclusions on cross examination under oath as follows:

"I said that, \* \* \* the findings of an acute nephrosis, associated with central necrosis of the liver in an individual who gives a history of having worked with carbon tetrachloride strongly suggests that we are dealing with a case of acute nephrosis and its complications, due to carbon tetrachloride poisoning;"

Dr. Rosenthal further testified:

"The reason of my opinion was that after performing the autopsy and finding the changes of nephrosis and necrosis of the liver which I have always associated with some acute and rather violent poisoning; that when I was given the additional history of the subject—having worked with this chemical (carbon tetrachloride), I deduced, as is customary in our work, that the subject was most likely, that these changes were most likely the result of the poison he had been working with."

Concerning the time element the doctor testified:

Q. "And it is your firm opinion that the changes you found in the autopsy and the anatomical condition were produced by this carbon tetrachloride?"

A. "It is my firm opinion the changes found could have been produced by carbon tetrachloride."

Q. "And reasonably were?"

A. "I think probably they were."

The petitioners seek to have us overthrow the findings of the Commission on some six principal grounds:

(a) That the pathologist did not actually find carbon tetrachloride poison in the body of the deceased when performing the autopsy.

(b) That the testimony of Doctors Armbruster and Rosenthal was conjectural, and that each relied upon the testimony of the other as a basis for his conclusion as to the cause of death.

(c) That other poisons could have caused the same condition found in the body of the deceased by the pathologist.

(d) That certain symptoms of carbon tetrachloride poisoning, such as jaundice and irritation of the eyes, nose, throat and lungs, were not present in this case.

(e) That according to the authorities there was insufficient carbon tetrachloride in the deceased's possession (some four or five ounces in all) to have diffused the liters of space in the known cubical content of this room to such an extent, even if it had all been spilled at once,

to have made the air in the room even highly dangerous, let alone furnish a lethal dose.

(f) That their medical witnesses, Doctors A. M. Tuthill and Norman Ross, both of whom are eminent in their profession, disagreed with the diagnosis as to cause of the deceased's death, of the other doctors called by the applicant. The two doctors named above readily admitted that neither of them were trained pathologists. Their testimony was largely confined to opinion evidence.

We have carefully considered each of these objections and do not consider that any or all of them are fatal to the findings and conclusion of the Commission. In general they merely raise a conflict in the evidence.

We have repeatedly held that if there is any conflict of medical or other testimony, such conflict will be resolved in favor of the findings of the Industrial Commission. *Stephens v. Miami Copper Co., et al*, 59 Ariz. 528, 130 Pac. (2d) 507; *Caekos v. Stanley Fruit Co.*, 55 Ariz. 72, 98 Pac. (2d) 471.

Every presumption is in favor of the award of the Commission and it is only in cases where there is no reasonable evidence to support its findings that the award will be set aside. We examine this evidence, not as triers of facts, but for the purpose of determining whether the record contains substantial evidence supporting their findings or conclusions.

Another well settled rule in this jurisdiction is that in compensation proceedings, if evidence is such that reasonable inference may be drawn therefrom either way, the Supreme Court must sustain award of the commission. We cite but two of our many cases so holding. *Vest v. Phoenix Motor Co., et al*, 50 Ariz. 137, 69 Pac. (2d) 795; *Young, et al, v. Hodgman and Mac Vicar*, et al, 42 Ariz. 370, 26 Pac. (2d) 355.

It is, of course, the law that when an application for death benefits is made, it is incumbent upon the claimant to show affirmatively, to the satisfaction of the Commission, sitting as triers of the fact, (a) that there was an accident arising out of and in the course of employment; and (b) that the deceased was injured thereby, and (c) that the injury was the cause of death; *David v. Industrial Commission*, 46 Ariz. 169, 49 Pac. (2d) 394.

It would seem to be conclusively established by all of the medical testimony in the case that the deceased came to his death as a result of some toxic substance getting into his system. Furthermore, it was a case of acute and not chronic poisoning. It appears that while findings Nos. 3 and 4, as shown by the pathologist's report, supra, might occur under many circumstances and from various causes, yet findings Nos. 1 and 2 only appear as a result of an acute toxic condition. While the doctors might conjecture as to what particular poisonous substance caused death, the Commission evidently

agreed with Dr. Armbruster, the family physician, who said "there was no sense in going into something foreign because Mitchell wasn't using anything but carbon tetrachloride." It also appears from the record that the deceased was probably more susceptible to the effects of carbon tetrachloride than others. Dr. Rosenthal termed this an "idiosyncrasy to different drugs". According to the doctors, had the deceased been highly allergic, or had an individual toxicity, to this drug, its first use would have produced prompt fatal results. There is nothing to show that the deceased was aware of his sensitiveness to this substance which he was constantly using.

We hold that there was ample evidence before the Commission to warrant their implied findings that the deceased came to his death as a result of inhaling carbon tetrachloride fumes between the 24th to the 30th day of December, 1942, which occurred in the course of his employment by petitioner.

It is suggested in the briefs that the death of the deceased resulted from an occupational disease and hence was not compensable under our Workmen's Compensation Law. Such a contention is untenable for the reason that, while it is true an occupational disease is not compensable under the Workmen's Compensation Law, such a disease is one which is due wholly to causes and conditions which are normal and constantly present and characteristic of the particular occupation; that is those things which science and industry have not yet learned how to eliminate, such as silicosis or chronic lead poisoning and similar diseases. It is a disease acquired by a slow, gradual process in the ordinary course of employment and as an incident thereto. *Industrial Commission v. Roth*, 98 Ohio St. 34, 120 N. E. 172; 6 A.L.R. 1463; *Ramsay v. Sullivan Mining Co.*, 51 Ida. 366, 6 Pac. (2d) 856; *Ross v. Ross*, 184 Okla. 626, 89 Pac. (2d) 338.

More specifically the injury under consideration in this case, wholly fails to meet the requirements laid down in the recently enacted Arizona Occupational Disease Disability Law, Chap. 26, Laws 1943. See also *Industrial Commission v. Frohmiller*, (Ariz.) 140 Pac. (2d) 219.

It appears that petitioner, and other companies similarly engaged, issue to their recondition repair men carbon tetrachloride in small quantities for use in their work. Other than deceased's fatal experience, injury arising therefrom has been negligible; in fact, no previous instance of injury to those thus engaged in this industry was cited by either party. The record in this case shows that the ordinary use of carbon tetrachloride in this business is not only not fatal but is harmless when properly used. The fatality here involved was undoubtedly caused by the inability of the de-



ceased on the Goodyear job, for the reasons heretofore stated, to comply with the petitioner's instructions given to its employees as to the use of carbon tetrachloride, "to use only in well ventilated places, and to avoid inhaling fumes." Evidently too much is not known about this rare type of poisoning, but it is recognized that the use of carbon tetrachloride is an industrial hazard. Major General H. L. Gilchrist, Chief of the U. S. Chemical Warfare Service states: "We are not yet ready, definitely, to announce the lethal concentration for single exposures". Bulletin 564, U. S. Bureau of Labor Statistics, pages 176-177.

Having concluded that the first question stated at the beginning of this opinion must be answered in the affirmative, we turn now to the more vexatious and perplexing question as to whether the deceased's death was the result of an injury by accident arising out of and in the course of his employment.

Such variable definitions have been given by the appellate courts of the United States to the words "injury by accident", "accidental injury", etc., and the application of such definitions to given cases or states of fact have resulted in such a divergence of concept and interpretation that the decisions cannot be harmonized or brought into unison. This is partly due to a difference in the statutes involved and partly because of a conflict of judicial thought and reasoning. Certainly the writer of this opinion has no intention of undertaking the herculean and impossible task of attempting to reconcile the various decisions.

Most of the states having comparable Workmen's Compensation statutes to Arizona, patterned their law after the Act passed by the British Parliament, and the appellate courts of the great majority of these states, including our neighboring states of New Mexico, Colorado, Utah and Idaho, have adopted and followed the liberal English rule announced by the House of Lords in the leading case of *Fenton v. Tharley* (1903) A. C. 443. From the jurisdictions adopting this rule numerous cases might be cited that would support the ruling of the Industrial Commission in the instant case. To show this trend we cite but a few typical cases, the facts therein may not be comparable, but the liberal interpretation is unmistakable. *McNeely v. Carolina Asbestos Company*, 206 N. C. 568, 174 S. E. 500; *Webb v. New Mexico Pub. Co.*, (N. M.) 141 Uac. (2d) 333; *Tintic Milling Co. v. Industrial Commission*, 60 Utah 14, 206 Pac. 278; *U. S. F. & G. Co. v. Industrial Commission*, 76 Colo. 241, 230 Pac. 624; *Industrial Commission v. Roth*, (Ohio) supra; *Sullivan Mining Co. v. Ashenbach*, 33 Fed. (2d) 1; *Tri-State Contractors, Inc., et al v. Alt-house*, 166 Okla. 296, 27 Pac. (2d) 1041; *Dee Memorial Hospital Assn. v. Industrial Commission*, (Utah) 138 Pac. (2d) 233.

We have pointed out many times that the Workmen's Compensation Law of Arizona is based in the main upon that of Utah and the Supreme Court of Utah has said that their law was patterned after the Ohio statute. *Industrial Commission v. Frohmiller*, supra. In our decision in the leading case of *Pierce v. Phelps Dodge Corporation*, 42 Ariz. 436, 26 Pac. (2d) 1017, we came to a parting of the ways with the Utah court and the other courts following the English rule, on the vital question of the meaning of "injured \* \* \* by accident" and particularly of the word "accident". We there held in effect that there must be some *sudden or instantaneous event or occurrence* which, taken by itself can be recognized as an accident, and then that the injury must be shown to have followed as a consequence from that specific event. Coupled with this was the implication that there must always be an *external act or occurrence*, usually one of *violence*, which caused the injury or death.

This somewhat restricted and rather narrow rule announced in the *Pierce* case has been praised and followed outside of Arizona in the following cases: *Booke v. Workmen's Compensation Bureau*, 70 N. D. 714, 297 N. W. 779; *Stanton v. Minneapolis Ry. Co.*, 195 Minn. 457, 263 N. W. 433; *Demagalski v. State Industrial Accident Comm.*, 151 Ore. 251, 47 Pac. (2d) 947.

On the other hand it has been cited and criticized, but not followed, in the following cases: *Smith, et al. v. McHan Hardware Co.*, 56 Ida. 43, 48 Pac. (2d) 1102; *McCormick Lumber Co. et al. v. Department of Labor and Industries*, et al, 7 Wash. (2d) 40; 108 Pac. (2d) 807; *Stevenson v. Lee Moor Contracting Co.*, 45 N. M. 354, 115 Pac. (2d) 342; *Webb v. New Mexico Publishing Co.* (N. M.) 141 Pac. (2d) 333; *Christensen v. Dysart*, et al. 42 N. M. 107, 76 Pac. (2d) 1; *Meyer v. Reettele*, 64 S. D. 36, 264 N. W. 191; *Lumberman's Mutual Casualty Co. v. Griggs*, 190 Ga. 200, 9 S. E. (2d) 84.

Our holding in the *Pierce* case is, however, the law in Arizona, unless we see fit to overrule or modify the same. But it requires no citation of authority for the proposition that this court is not bound by its former decisions, unless the declaration of principles in such former adjudicated cases commend themselves by their essential soundness.

Petitioners urge that the award made in this case can not stand if we strictly adhere to the accident rule laid down in the *Pierce* case. This is doubtless true. While we agree that a correct ultimate conclusion was reached in disposing of the *Pierce* case, under the facts there shown yet we now feel that probably too little weight was given to the peculiar and appropriate meaning in the law (Sec. 1-103, sub. 1, A.C.A. 1939) of the phrases such as "injured—by accident" and "accident" appearing in



our compensation law, which phrases had a well defined and well understood meaning at the time of the enactment of our Workmen's Compensation Act.

Furthermore it appears that in the *Pierce* case we did not discuss or consider, probably because it was not called to our attention, the phrasing of the Constitutional mandate to enact a Workmen's Compensation law. Certainly the legislative intent can best be gleaned by reference to Section 8, Article 18 of the Arizona Constitution, which provides that "compensation shall be . . . paid . . . if in the course of such employment personal injury to or death of any such workman from any accident arising out of, and in the course of, such employment, is caused in whole, or in part, or is contributed to, by a necessary risk or danger of such employment, or a necessary risk or danger inherent in the nature thereof . . .".

It will be noted that the italicized part of the Constitution just quoted is broader and more comprehensive than the legislative enactment appearing under Section 56-936, A.C.A. 1939. A construction of the latter must be governed by the constitutional provision.

Applying these principles to the instant case it is readily apparent that the poisoning of the deceased, which caused his death, was "caused in whole, or in part, or was contributed to, by a necessary risk or danger of such employment".

While accidental injuries are usually sudden happenings and are caused by some violent or external means such as a traumatic injury, not all accidents fall into such a mold, or such a straight-jacketed pattern. There is nothing in our statutory law, Sec. 56-931, (formerly Sec. 1421 R. C. 1928) that says that an industrial accident must be an instantaneous happening or that violence need be a part thereof in order for a resulting injury to be compensable.

In the exceptional cases, such as the one now under review, the injury suffered was unquestionably accidental, though the precise time of the beginning may have been uncertain. An injury may be gradual and progressive and not immediately discoverable. Here the poisonous effect was undoubtedly cumulative over the short period of time that the deceased was working at the Goodyear plant. The conditions under which the deceased was working were unusual and abnormal for the class of work he was doing. He was in a confined space, in an inadequately ventilated room, with a highly volatile, poisonous chemical, which should only have been used in a well ventilated room. He breathed the fumes and was poisoned.

We know that the employer did not wilfully maintain the conditions there found to exist, or intentionally cause the poisoning and death. The deceased, although he apparently recognized that the fumes were dangerous, and returned to work in disregard of the doctor's or-

der, cannot be charged with deliberately committing suicide. No one expected, anticipated or looked for the poisoning and death to deceased. The result was undesigned and unexpected. It was therefore an event which took place without one's foresight or expectation, and hence a fortuitous happening.

We know that the employer did not wilfully said in the *Pierce* case to the contrary notwithstanding, that there was an injury by accident in this case within the meaning of the Arizona Law, as now interpreted, in that the inhalation by the deceased of the fumes from the use of the poisonous carbon tetrachloride, under the circumstances heretofore stated, produced effects that were not intended, foreseen or expected. This was an unlooked for mishap, an unexpected, unusual and extraordinary event not reasonably contemplated as a part of normal conditions of employment.

In making this ruling we disavow, in advance, any intention of letting down all the bars in these Industrial cases; we have here removed only those bars which prevented justice being done in this case under a common-sense interpretation of the constitution and statutory law of this State. The provisions of the Workmen's Compensation Act are remedial in character and should be construed liberally in favor of the workmen. *Ocean Acc. & Guar. Corp. v. Industrial Commission*, 32 Ariz. 265, 257 Pac. 641; *Butler v. Industrial Commission* 50 Ariz. 516, 73 Pac. (2d) 703. The fund is created to protect the workman; but it is limited to injuries by accident arising out of and in the course of the employment. It is not a health or accident insurance fund. The deceased was an industrial casualty, and his dependents are entitled to death benefits. We confine ourselves to a determination of the law as applied to the facts shown in this case.

Award affirmed.

#### SAN FRANCISCO HEART COMMITTEE

Dr. Chester S. Keefer of Boston will be one of the speakers at the San Francisco Heart Committee's Fifteenth Annual Postgraduate Symposium on Heart Disease which will be held this year October 26, 27, and 28. Other guest speakers will be Dr. Maxwell M. Wintrobe, Professor of Medicine, Utah University Medical School; Dr. S. J. McLendon of San Diego, Dr. William Gordon of the United States Public Health Service, and others to be announced later. More than twenty San Francisco physicians especially interested in cardiovascular disease will participate in the Symposium. Rheumatic fever will receive special emphasis this year.

The sessions will open Thursday, October 26 at the University of California School of Medicine. The program for Friday, October 27 will be presented at Stanford University School of Medicine and at the San Francisco Hospital. A dinner meeting has been arranged for the evening of the 27th at the St. Francis Hotel. Saturday morning's pro-

(Continued on Page 296)

# ORGANIZATION SECTION

DAN L. MAHONEY, M. D., President

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## PRESIDENT'S MESSAGE

The summer has been given over to vacations for the most part. We are pleased to announce that committee personnel are now complete, the roster appearing in the ORGANIZATION SECTION of each issue of this *Journal*. You are familiar by now with the By-law provisions for setting up committees under the headings of SCIENTIFIC and NON-SCIENTIFIC. One of the best features of committee organization is that a member takes appointment for a term of three years, so that there are two experienced hold-over members each year and one new member, as personnel is set at a membership of three except in two or three instances. Due to so many of our members going into service, we have experienced a little difficulty in securing permanent committee personnel. It appears at present as if the Association now has a full staff of committees with personnel that will be on the job for the full tenure of office.

We ask that you look over the list of committees, note the title of each which indicates its scope of work, then lay before each one any medical problem in your community that you

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feel needs the direct attention of the committee. If you have any problem on Industrial Health, Social Diseases, Tuberculosis, Crippled Children, Maternal and Child Health, or others of scientific nature, you will note that the Association has a committee to aid in working out a solution. The same applies to Non-Scientific matters such as Medical Economics, Public Health, State Health Relations and the like. The Committee on State Health Relations, for example, is to assist the various state institutions such as the State Hospital, Boys' Industrial School, Pioneers' Home, State Prison, School for the Blind and the like, in their medical problems. The Governor has said that he plans to call on this committee frequently for such aid as it may give in institutional medical problems. Dr. F. W. Butler of Safford has recently been appointed by the Governor to serve a five year term on the State Board for Juvenile Institutions, this board directs the activities of institutions caring for delinquent boys and girls of the state. Dr. Butler was selected from the list provided the Governor, in conformance with the law, the Council approving the list of three names submitted for consideration.

The Association committees are all active as each has a specific job of work to do. Your society will hear from each committee during the course of its activities and you are urged to respond to any requests committee may make for society participation and endorsement. The Council approves all committee programs before they are put into action. The Committee on Scientific Education and Post Graduate Activities, now that we have a complete personnel once again, will draft its program in the near future and give the societies something definite to promote for scientific study and advancement.

This issue of the *Journal* also carries a roster of the association membership. While we have lost a considerable number to the armed forces, we have also picked up new members from among those coming into the state to locate. We are a small association, numerically, but an active one in that our component societies and members are always interested and active in state and national organization work.

DAN L. MAHONEY, M. D.,  
President.

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Coolidge
- McCracken, Paul W.  
Phoenix
- McIntyre, A. J.  
Phoenix
- McKeown, H. J.  
Phoenix
- McKhann, Geo. G.  
Phoenix
- McMartin, H. L.  
Phoenix
- McVay, L. Clark  
Phoenix
- Meason, Jas. M.  
Chandler
- Medigovich, D. V.  
Phoenix
- Melton, B. L.  
Phoenix
- Milloy, Frank J.  
Phoenix
- Mills, C. W.  
Tucson
- Mills, Harlan P.  
Phoenix
- Moon, O. B. \*\*\*  
Bisbee
- Moore, Jas. R.  
Phoenix
- Moran, Tressa R.  
Phoenix
- Morton, Wm. G.  
Winslow
- Nagoda, Ed J.  
Tucson
- Nelson, D. E.  
Safford
- Nevins, C. R.  
Casa Grande
- Noice, Russell R.  
Miami
- Oatway, Wm. H.  
Tucson
- Ochs, Melvin L.  
Jerome
- Ohl, Howard Jay  
Phoenix
- Omer, Joy A.  
Tucson
- Ovens, Jas. M.  
Phoenix
- Palmer, Chas. B.  
Phoenix
- Palmer, E. Payne  
Phoenix
- Palmer, Ralph F.  
Phoenix
- Parrish, F. W.  
Bowie
- Patterson, C. E.  
Tucson
- Patterson, John H.  
Phoenix
- Penn, R. L.  
Avondale
- Pennington, John W.  
Phoenix
- Phillips, Earle W.  
Phoenix
- Phillips, Robert T.  
Phoenix
- Phillips, Wm. A.  
Yuma
- Ploussard, Chas. N.  
Phoenix
- Podolsky, A. I.  
Yuma
- Pohle, Ernest  
Tempe
- Presson, Virgil G.  
Tucson
- Purcell, Geo. W.  
Tucson
- Rafferty, T. N.  
Phoenix
- Randolph, Howell S.  
Phoenix
- Randolph, Victor S.  
Phoenix
- Raymond, R. O.  
Flagstaff
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Whipple
- Reese, F. L.  
Phoenix
- Rice, Hal W.  
Bisbee
- Robb, Mayo  
Phoenix
- Rogers, Geo. K.  
Phoenix
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Tempe
- Rosenthal, Maurice  
Phoenix
- Ross, Norman A.  
Phoenix
- Rubel, Geo. C.  
Buckeye
- Rudolph, Royal W.  
Tucson
- Running, E. Henry  
Phoenix
- Ryerson, Paul M.  
Phoenix
- Saxe, Louis J.  
Phoenix
- Schoffman, Wm. F.  
Phoenix
- Schultz, Wm. M.  
Tucson
- Schwartz, W. A.  
Phoenix
- Scott, A. R.  
Flagstaff
- Sechrist, Chas. W.  
Flagstaff
- Sharp, Floyd B.  
Phoenix
- Sharp, W. S.  
Mesa
- Shearer, J. E.  
Phoenix
- Shelley, A. A.  
Phoenix
- Sherrill, W. Paul  
Phoenix
- Smelker, V. A.  
Tucson
- Smith, Chas. S.  
Nogales
- Smith, R. K.  
Tucson
- Smith, Willard \*\*\*  
Phoenix
- Snyder, Bertrand L.  
Phoenix
- Spearman, F. W.  
Williams
- Sprinkle, Paul D.  
Winslow
- Stratton, J. Newton  
Safford
- Stratton, Robt. A.  
Morenci
- Stevens, Robt. H.  
Phoenix
- Steward, Gus B.  
Coolidge
- Stroud, Reginald J.  
Tempe
- Sturgess, J. Hubert\*  
Yuma
- Sult, Chas. W., Sr.  
Phoenix
- Swackhamer, C. R.  
Superior
- Swetnam, C. R. K.  
Prescott
- Tappan, Vivian  
Tucson
- Taylor, John T.  
Cottonwood
- Thomas, Chas. A.  
Tucson
- \* Moved from State  
\*\* Associate Member  
\*\*\* Deceased
- Thomas, Naugle K.  
Tucson
- Thompson, A. B.  
Tucson
- Tompkins, L. M.  
Gilbert
- Townsend, S. D.  
Tucson
- Tucker, J. B.  
Florence
- Tuthill, Alexander M.  
Phoenix
- Utzinger, Otto E.  
Ray
- Walker, Glen H.  
Coolidge
- Ward, R. Leslie  
Buckeye
- Watkins, Evelyn G.  
Tucson
- Watkins, W. Warner  
Phoenix
- Watson, Samuel H.  
Tucson
- Webster, Clara S.  
Tucson
- Welton, P. C.  
Tucson
- West, O. C.  
Phoenix
- White, Toler R.  
Kingman
- Whiting, Spencer D.  
Phoenix
- Williams, Marguerite  
Tucson
- Wills, E. C.  
Phoenix
- Wilson, C. L.  
Yuma
- Wilson, John C.  
Willcox
- Wilson, R. A.  
Tucson
- Woern, Wm. H.  
Phoenix
- Woodard, J. H.  
Tucson
- Wright, Morton G.  
Winslow
- Wyatt, B. L.  
Tucson
- Yount, Clarence E.,  
Prescott
- Yount, Florence B.  
Prescott
- Zemsky, Boris  
Tucson
- Zinn, P. P.  
Bisbee

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- Armbruster, A. Carl  
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Phoenix
- Barker, C. J., Jr.  
Phoenix
- Barnes, H. A.  
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- Bendheim, O. L.  
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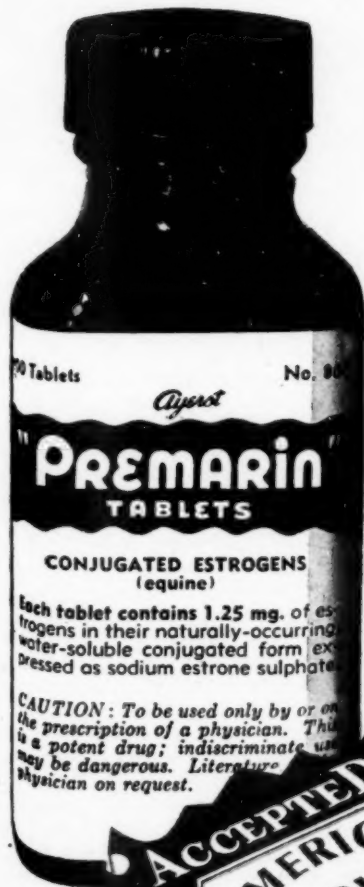
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| Brainard, H. H.<br>Tucson         | Kimball, Albert P.<br>Yuma        |
| Bregman, E. H.<br>Tombstone       | Kimball, Robt. M.<br>Yuma         |
| Brinkerhoff, D. E.†<br>Phoenix    | Kirmse, Alvin<br>Tucson           |
| Brizzolara, C. M.<br>Douglas      | Kitt, W. Stanley<br>Tucson        |
| Brown, Preston T.<br>Phoenix      | Kittredge, D. W. Jr.<br>Flagstaff |
|                                   | Knight, F. W.<br>Safford          |
| Callander, R. J.<br>Tucson        | Kober, Leslie R.<br>Phoenix       |
| Carrell, W. D.<br>Tucson          | Koehn, Carl L.<br>Goldroad        |
| Condell, Lyle A.<br>Safford       | Koerber, F. L.<br>Phoenix         |
| Condon, D. J.<br>Phoenix          | Kohl, Harold W.<br>Tucson         |
| Craig, Carlos C.<br>Phoenix       |                                   |
| Creighton, Carol C.<br>Flagstaff  | Lentz, Jos. S.<br>Phoenix         |
|                                   | Lewis, Donald B.<br>Tucson        |
| DePinto, A. J.<br>Phoenix         | Linton, C. S.<br>Tucson           |
| Dysart, Palmer<br>Phoenix         | Littlefield, Jesse B.<br>Tucson   |
|                                   |                                   |
| Engelder, A. E.<br>Tucson         | Mandel, M. M.<br>Tucson           |
|                                   | Matts, Robt. M.<br>Yuma           |
| Farness, O. J.<br>Tucson          | Maury, F. H.<br>Tucson            |
| Fife, Karl L.<br>Duncan           | McNally, Jos. P.<br>Prescott      |
| Fillmore, A. J.<br>Mesa           | Merrill, M. W.<br>Phoenix         |
| Flood, Clyde E.<br>Tucson         | Mikell, John S.<br>Tucson         |
| Francis, J. Donald<br>Tucson      | Mills, C. Selby<br>Phoenix        |
|                                   | Montgomery, R. E.<br>Douglas      |
| Graham, D. E.<br>Douglas          |                                   |
| Greer, Joseph M.<br>Phoenix       | Neff, B. L.<br>Mesa               |
| Gunter, Manning<br>Globe          | Ness, A. R.<br>Miami              |
| Gwinn, F. W.<br>Yuma              | Noon, Z. B.<br>Nogales            |
|                                   | Nugent, A. G.<br>Douglas          |
| Haines, R. S.<br>Phoenix          |                                   |
| Hall, Norman D.<br>Phoenix        | O'Neill, Jas. T.<br>Coolidge      |
| Hastings, R. E.<br>Tucson         |                                   |
| Hein, Walter F.<br>Williams       | Palmer, E. Payne, Jr.<br>Phoenix  |
| Herzberg, Benjamin<br>Phoenix     | Palmer, Maxwell R.<br>Tucson      |
| Hewitt, W. Roy<br>Tucson          | Palmer, Paul V.<br>Phoenix        |
| Hicks, R. A.<br>Tucson            | Perkins, F. P.<br>Tucson          |
| Holbrook, William P.<br>Tucson    | Peterson, K. E.<br>Phoenix        |
| Hurianek, Z. A. (fem.)<br>Phoenix | Piepergerdes, C. C.<br>Bisbee     |
| Hussong, R. W.<br>Phoenix         | Pohle, C. L.<br>Chandler          |
|                                   | Polson, Donald A.<br>Phoenix      |
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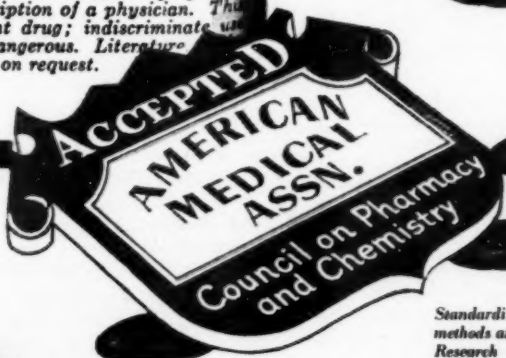
● "Premarin" is highly potent; even severe and well-established cases usually show a satisfactory response to treatment.

● Although highly potent, "Premarin" is exceptionally well tolerated; unpleasant side effects are seldom noted.

● "Premarin" is a natural estrogen . . . conjugated estrogens (equine).

● "Premarin" therapy conserves the physician's time; office consultations are kept at minimum.

● "Premarin" is accepted by the A.M.A. Council on Pharmacy and Chemistry.



Standardized by colorimetric and biological methods and supplied with the approval of The Research Institute of Endocrinology, McGill University, under direction of Dr. J. B. Collip.



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Ricker, John Phoenix	Thoeny, O. W. Phoenix
Saba, Joseph Bisbee	Thomas, John Wix Phoenix
Sarlin, C. N. Tucson	Thompson, H. E. Tucson
Schnabel, G. P. Phoenix	Thompson, Hugh C., Jr. Tucson
Secrist, D. L. Tucson	Truman, Geo. C. Mesa
Shultz, Wm. G. Tucson	Tucker, W. P. Superior
Shupe, Reed Phoenix	Tuveson, L. L. Phoenix
Sickler, J. R. Tucson	Ure, W. G. Tucson
Smith, Leslie B. Phoenix	Van Epps, Chas. E. Phoenix
Smith, William J. Phoenix	Wallar, J. Lloyd Yuma
Southworth, Harry T. Prescott	Walsh, Jas. S. Douglas
Stacey, John W. Tucson	Warrenburg, C. B. Phoenix
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Steen, W. B. Tucson	Wheeler, Norman O. Globe
Steward, B. L. Florence	Williams, Henry G. Phoenix
Storts, B. P. Tucson	Williams, Onie O. Phoenix
Stump, Robt. M. Phoenix	Woodman, Thos. W. Phoenix
Sult, Chas. W., Jr. Phoenix	Yount, C. E., Jr. Prescott

† Returned to practice.

## Clinical Pathological Conferences

### GOOD SAMARITAN HOSPITAL CASE—JULY, 1944

Patient was a 45-year-old white male, who was admitted on February 6, 1944, complaining of generalized neuritic and arthritic pains of threere months' duration. He also complained of generalized weakness, loss of weight and a slight impairment of vision during the last several months. He had been apparently well until four years ago when he had an attack of acute pain in the left leg, which was said to be due to phlebitis. A short time after that, he developed pain in the right leg and hip, which was similarly diagnosed. An x-ray at this time revealed a bone tumor of the right femur. A

diagnosis of primary bone tumor was made at the Mayo Clinic, for which he was treated. A year before admission to this hospital, patient began to complain of pain in the right shoulder and arm. This pain was transient and migratory in character. For the past two months the patient had complained of a constant numbness of the lower lip and chin. During the past three or four weeks, he had a low grade, intermittent fever. His general course was down-hill, with loss of weight and strength.

Physical examination revealed a large frame, robust type of subject who was over-weight and presented a plethoric facies. There was a slight droopiness of both eyelids. The pupils were dilated and fixed. The eye grounds showed no pathological changes of significance at the first examination. The teeth were in a good state of preservation, and showed nothing of clinical significance. The chest presented a barrel-shaped thorax. The lung fields were essentially negative. The heart showed nothing of significance. The abdomen revealed no masses or areas of tenderness. The extremities were essentially normal, except for an old 4-inch scar in the mid portion of the right thigh. The neurological examination presented: 1. Suggestive drooping of eyelids; 2. Dilated, fixed pupils, left larger than right, definite impairment vision left eye; 3. Paresthesia of the anterior mandibular region; 4. Tendon reflexes were depressed and somewhat erratic; 5. Abdominal and cremasteric reflexes were absent.

#### Laboratory Data

Routine agglutinations were negative. Spinal puncture revealed a normal pressure, with clear fluid; microscopic examination was negative; Pandy's was positive. Stool examination was negative for ova, cysts and parasites. Culture was negative for pathogenic organisms. Blood serology and spinal fluid serology was negative. Non-protein nitrogen was 28; sugar was 75. Red blood count was 4,610,000; leukocytes, 15,600; hemoglobin, 13.8 grams. Differential count was normal. The urine showed a specific gravity of 1015, and a faint trace of albumin; there were two or three white blood cells per high power field, and a few finely granular casts. Sedimentation rate was 27 mm. at the end of an hour by the Cutler method. X-ray examination of the sella tursica showed no evidence of increase in size or other abnormality. X-ray examination of the gallbladder showed a non-functioning gallbladder. There were no visible metastatic bone lesions in areas investigated by x-ray examination.

#### Progress

Several weeks before he expired, patient was given an eye examination which revealed a retinal separation involving the outer half of the retina of the left eye. There was no evidence of vision in this eye. About two weeks before he died, patient became markedly jaundiced.



FOR SOME WOMEN the climacteric is practically uneventful except for the cessation of menstrual function. To others the cessation of ovarian activity becomes a crisis to themselves and their families.

One of the valued contributions of endocrinology was the discovery and isolation of potent estrogens and their usefulness in alleviating the distressing symptoms of the menopause.

Within the last five years the natural estrogens have decreased in cost while orally administered diethylstilbestrol costing only one or two cents a day has brought estrogenic therapy within the reach of every woman.

The Squibb Laboratories supply the natural estrogens Amniotin, in a variety of dosage forms for use orally, intravaginally or by hypodermic injection. The

present price of Amniotin in Oil in capsules is the lowest in history; for injection, Amniotin in Oil in the vial packages makes the cost per dose relatively inexpensive and enables the physician to vary the dosage to fit the patient's requirements.

Diethylstilbestrol Squibb is available in a like variety of dosage forms except that for oral administration it is supplied in tablets rather than capsules. Reports in the literature indicate that, where excessive dosage is avoided, many patients acquire a tolerance to the drug and are able to take it without discomfort.

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**BUY AN EXTRA WAR BOND . . . — FOR VICTORY**

The patient continued a gradual down-hill course, and expired several weeks after his second admission to the hospital.

**DR. W. W. WATKINS:** This patient was first seen on Feb. 1, 1940, being referred by Dr. Greer with the history that patient had just returned from the Mayo Clinic, where some type of bone tumor involving the right femur had been explored and a diagnosis of bone sarcoma made. Dr. Watkins' report was that a destructive medullary bone lesion was shown, which has eroded the cortex, but the x-ray appearances were not conclusive as to whether this is osteogenic sarcoma or Ewing tumor, and further data from the Mayo Clinic was secured.

The previous history given by Dr. Greer was that this man and his family had been his patients for years, but Dr. Greer was not consulted about the so-called "rheumatism" in this thigh which developed in the Fall of 1939. This man consulted another doctor in Phoenix and was treated by him for two or three months, no x-ray films being made. Patient then went to Mayo Clinic, on his own initiative, where x-ray films were made, bone tumor found and this was explored, a specimen being taken for biopsy. The pathological report was grade 4 hemangio-endothelioma or Ewing sarcoma. Since this growth has a very serious prognosis, but is radiosensitive, x-ray therapy usually

gives as good results as surgery. He was therefore advised to have x-ray therapy, one series being given at the Mayo Clinic with advice to have this treatment continued in Phoenix. His treatments were completed at the Clinic about Jan. 25th.

Patient started the second series at Pathological Laboratory on Feb. 25, 1940, using somewhat higher voltage than was given at the Mayo Clinic.

Film of 2/2/40 shows the amount of bone destruction at that time.

Series of treatments were completed March 4, 1940. These produced a very satisfactory skin reaction.

Film of 5/4/40 shows a very satisfactory healing of this lesion. Patient was advised to have a short series of treatments once a month so as to maintain effect over a prolonged period of time. Such series were given in May, June and July, each of these series consisting of three reactions,—of 400 r each, or 1200 r for each series.

Film of 8/20/40 showed still further favorable change, although the soft tissue reaction was severe, consisting of a persistent edema which gradually changed to a contraction ring like a wide band about the thigh.

Film of 1/7/41 showed still further evidence of healing, with a small periosteal proliferation area where biopsy specimen had been removed.

In Nov., 1941, patient was seen by Dr. Merrill, the complaint being pain in back and a small tumor in the calf of right leg. Extensive x-ray survey was carried out without demonstrable evidence of bone lesions. A copy of our records and the x-ray films were sent to Mayo's for their advice. They agreed that visible bone metastases were not shown, but suspected that metastases might be taking place in soft tissues; since these were not sufficiently localized for x-ray therapy, they advised sending patient to Dr. Lawrence at Berkeley for treatment with irradiated phosphorus. The records were submitted to Dr. Lawrence, who did not recommend irradiated phosphorus for him. He wrote that all that could be expected had been accomplished in this case. Dr. Lawrence was very complimentary about the radiation technique used, and thought treatment with irradiated phosphorus would not add anything to this. He was of the opinion, as we had been and as the Mayo Clinic observers were when they refused to operate, that the opportunity to cure this radiosensitive tumor was lost by the delay in the outset, when months were wasted in treating this condition as "rheumatism". Dr. Lawrence thought that metastases which had already been planted at distant points, before any treatment was given, would eventually develop. That proved to be true.

In Feb., 1943, in June, 1943 and in Nov.,

Accident Hospital Sickness



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<b>\$5,000.00 ACCIDENTAL DEATH</b>	<b>For</b>
<b>\$25.00 weekly indemnity, accident and sickness</b>	<b>\$32.00</b>
	<b>PER YEAR</b>

<b>\$10,000.00 ACCIDENTAL DEATH</b>	<b>For</b>
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<b>\$15,000.00 ACCIDENTAL DEATH</b>	<b>For</b>
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	<b>PER YEAR</b>

**ALSO HOSPITAL EXPENSE FOR MEMBERS, WIVES AND CHILDREN**

*42 years under the same management*

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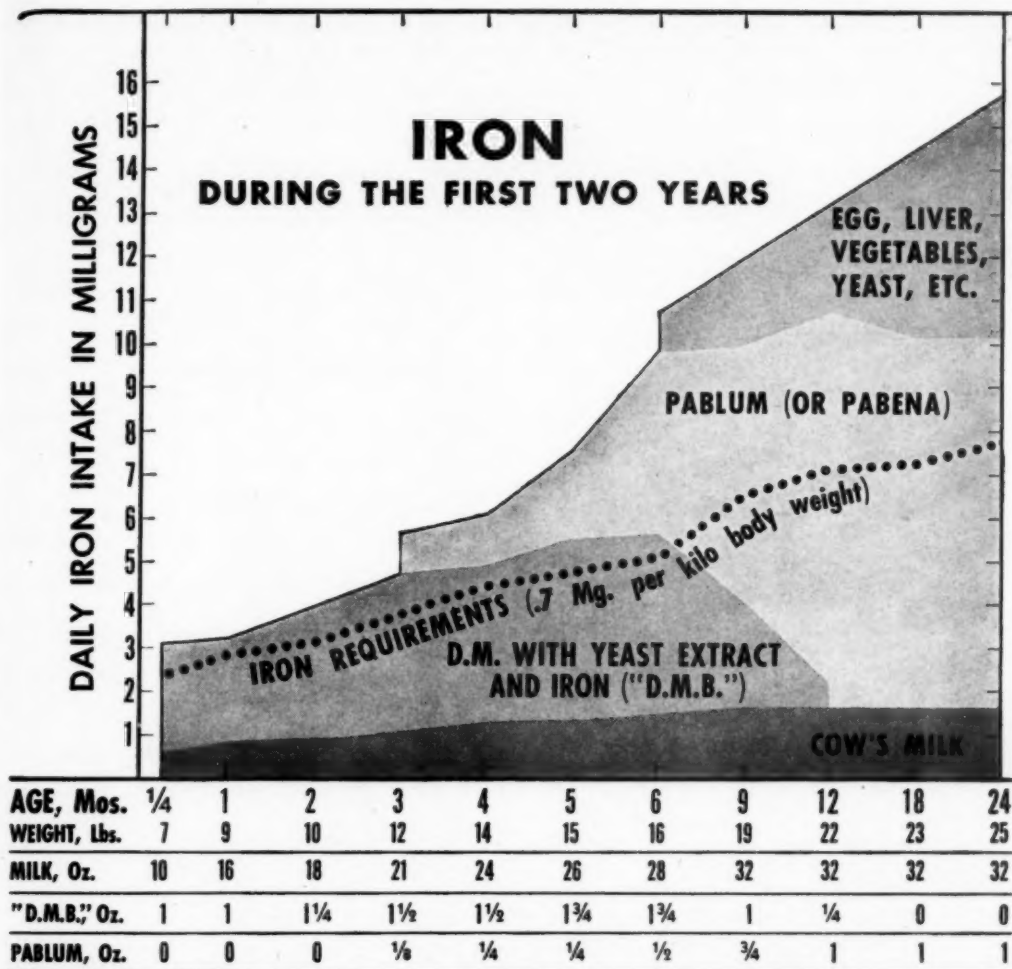
Disability need not be incurred in line of duty—benefits from the beginning day of disability.

*86c out of each \$1.00 gross income used for members' benefit*

**PHYSICIANS CASUALTY ASSOCIATION**  
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## IRON DURING THE FIRST TWO YEARS

During fetal life iron accumulates (in the form of hemoglobin) in the infant's body. After birth the hemoglobin frequently drops to 50% by the third month, especially in prematures. Neither breast milk nor cow's milk is capable of offsetting this loss, as they are deficient in iron. This chart shows that when the carbohydrate and cereal supplements contain iron, a sizeable margin of safety over the requirements can be maintained, not only during the important first six months, but throughout the first two years of life.

More iron than the calculated requirement is needed because some iron is not utilized. In rapidly growing, or poorly nourished infants, and in the presence of infection, the need for iron may be even greater than is indicated in this chart for normal infants.

**MEAD JOHNSON & COMPANY, Evansville 21, Ind., U.S.A.**

1943, x-ray examinations were made for Dr. Frissell. The lesion at the original site showed no further development, and in Nov., 1943, there was no x-ray evidence of lung metastases.

On 1/3/44, a skull examination was made for Dr. Saxe, who was under the impression that patient had myeloma. No bone changes were found in the skull.

A final survey of the gastro-intestinal tract and of several bond areas was made in this hospital in February, for Dr. Frissell, but no bony abnormalities were found other than in the right thigh.

The concensus of opinion during the general discussion was that this patient had a malignancy with general metastases throughout the body. The primary site of the malignancy was not decided upon.

#### *Pathologist's Report*

**DR. MAURICE ROSENTHAL:**—The body was that of a moderately well developed and nourished white male, approximately 50 years of age. The head was normal in shape and contour. The scalp was covered with light brownish, graying hair. There was no discharge from the nose, ears nor mouth. The eyelids and mouth were closed as a result of the embalming process. The mucous membranes were pale. There was no enlargement of the cervical, axillary and inguinal lymph nodes. There was a pitting edema of the ankles and lower extremities. There were no other abnormalities of the upper or lower extremities.

"Y"-shaped incision was made through the skin of the thorax and abdomen. The subcutaneous fat measured from  $\frac{1}{2}$  to 1 inch in thickness.

Upon entering the peritoneal cavity, the organs were found to be in their proper positions and relationships. The liver extended for a distance of three finger breadths below the costal margin in the mid-clavicular line. Upon removing the thoracic plate, string-like pleural adhesions were found between both upper lobes and the thoracic cage. There was no fluid in the pleural cavity. The organs were in their proper positions and relationships. The thoracic, abdominal and pelvic organs were removed, *entoto*.

The interior vena cava was normal, and contained no thrombi nor emboli. The aorta was stripped from its retroperitoneal attachment. The esophageal mucosa revealed several plaques of leukoplakia and a moderate degree of congestion.

The lungs, on sectioning, revealed a moderate degree of passive congestion and emphysema. Both lower lobes showed a slightly increased degree of passive congestion. No areas of lobular consolidation could be seen or palpated, grossly.

The heart was normal in size, shape, and in consistency. The myocardium of both ventricles

was normal, and all of the cardiac valves were essentially normal. The coronary ostia were patent, as were the coronary vessels. The aorta was elastic, and revealed only a few small, yellow, atheromatous plaques along the descending aorta.

Upon sectioning, the liver resembled a chronic passive congestion, and upon pressure an increased quantity of bile-stained material exuded from the cut surface.

The gallbladder was distended, as were the extra hepatic bile passages.

The pancreas was unusually hard and firm in consistency, and presented small focal, yellowish-gray nodules of infiltration. These were found in the head and body of the organ. The head was enlarged as a result of this process, and produced an obstruction of the common bile duct in the region of the papillae.

The spleen was slightly increased in size. The capsule was slightly thickened, and the cut surface showed no unusual gross pathological changes.

The adrenals were normal on cross sectioning.

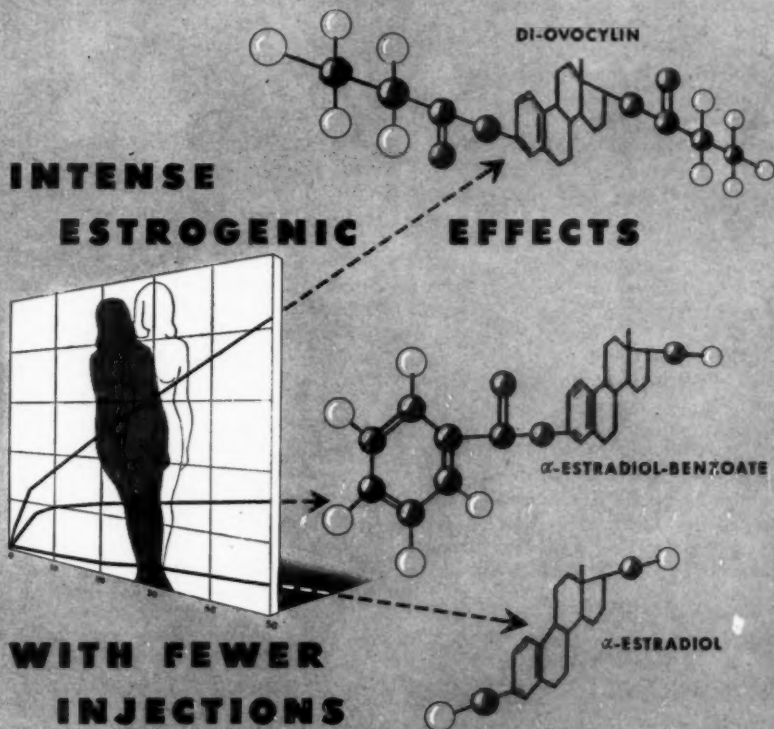
Both kidneys were enormously increased in size, and infiltrated by large round, yellowish-gray tumor masses, measuring 2 to 3 cm. in diameter. A few areas of normal kidney parenchyma were still discernible. The calices and pelvis were slightly dilated. The ureters were normal.

The bladder showed a moderate congestion of the mucosa.

The prostate was slightly increased in size, but unusually firm and hard in consistency. The cut surface presented a light yellowish-gray tumor of infiltration.

Upon examining the stomach, in the fundus and in the prepyloric region irregular-shaped broad masses of yellowish-gray tissue infiltrated and submucosa. These measured from 3 to 4 cm. in width and 5 to 8 cm. in length. There were no superficial ulcerations of the mucosa. The pylorus was normal, and the remaining gastro-intestinal tract showed only a moderate degree of congestion of the mucosa.

The scalp was incised from ear to ear and reflected backwards and forwards. Examination of the brain was limited to an occipital removal of the cranium. However, the calvarium was removed, and the dura appeared to be slightly thickened, but showed no other gross changes of pathological significance. The convolutions of the brain were normal in size and shape, and the subarachnoid space showed a slight excess of yellowish fluid. There were no areas of tumor infiltration in the meninges or cortex. Examination of the cerebral hemispheres showed no gross changes of pathological significance, nor did sectioning of the cerebellum reveal any changes of pathological significance. Sections through the pons, medulla



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and upper cervical cord revealed a few scattered areas of what appeared to be focal areas of degeneration. This may have been, however, only a focal edema. Sections through these areas were taken for microscopic study. The sympathetic ganglia along the cervical, thoracic and abdominal regions were examined, and the upper thoracic ganglia appeared to be increased in size, firm in consistency, and fibrous-like in appearance. Several of these were taken for microscopic study.

Several of the ribs showed irregular areas of increased density, apparently due to some infiltration. A section of rib was taken for microscopic study.

#### *Microscopic*

Sections through the prostate reveal focal proliferations of gland acini characteristic of an adenomatous hypertrophy of the prostate. In some of the sections, however, there is a diffuse cellular infiltration which is characterized by pleomorphism. With oil immersion, the cellular structure is seen comprised of endothelial cells, plasma cells, neutrophils and many erythroblasts. The erythroblastic series is quite prominent. The cells in many places are megakoblastic in character, and show mitosis. The cells are separated by a scant amount of eosinophilic-staining material. The general histologic characteristics are those of a mixed cell myeloma.

Sections through the kidneys show that the parenchyma has been replaced in many places by a cellular infiltration similar to that described above. The tubular epithelium shows degenerative changes. The interstitial vessels are congested, and the glomeruli and also infiltrated by similar tumor cells, and show degeneration and necrosis.

The capsule of the adrenal is also infiltrated by similar tumor cells. The cells of the cortex show degeneration.

Sections through the spleen reveal that the sinusoids are markedly dilated. There is a decrease of the lymphoid elements, and the pulp is infiltrated with numerous polynuclear cells and plasma cells.

The pancreas is also infiltrated by islands of tumor cells similar to those described above.

The liver shows marked cloudy swelling of the hepatic cells. Many of them show disintegration. The portal fields show a moderate infiltration with lymphocytes and plasma cells. Some of the hepatic cells show a fatty degeneration.

Sections through the lung show that the pleura is thickened as a result of a cellular infiltration comprised of tumor cells similar to those described above. The pleura is also edematous, and the vessels are congested. The pulmonary tissue adjoining the pleura is also infiltrated by tumor cells. The adjoining alveolar spaces

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<sup>1</sup> Biskind, G. R.: Proc. Soc. Exper. Biol. & Med. 43:259, 1940. Burrill, M. W. and Greene, R. R.: Endo. 31:73, 1942.

<sup>2</sup> Lissner, H. and Curtis, L. E.: J. Clin. Endo. 3:389, 1943.

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show that the capillaries are markedly congested. The infiltrations in the lung are focally distributed.

Sections through the wall of the stomach also reveal that the normal mucosa has been markedly infiltrated by similar tumor cells. The submucosa and muscularis are also infiltrated by islands of tumor cells. There is considerable edema and degeneration of the fibro-muscular elements. There is degeneration and necrosis of the epithelial cells.

The anterior lobe of the pituitary gland shows a cellular infiltration of the stroma with myeloid cells. There is degeneration and necrosis of some of the neighboring epithelial cells.

Sections through the pons, medulla and upper cervical cord show a slight cellular infiltration of tumor cells within dilated, perivascular lymph spaces. The nerve cells show degeneration. The reticular meshwork is widened, due to edema. The sympathetic ganglia show degeneration of the ganglion cells. No tumor infiltrations are found in the ganglia.

#### *Anatomical Diagnosis*

1. Carcinoma of prostate.
2. Carcinoma, metastatic, to pancreas, stomach, ribs and kidneys.
3. Emphysema, pulmonary.
4. Pleuritis, adhesive, chronic.
5. Obstruction of common bile duct.

6. Chronic inflammatory reaction of parasympathetic ganglia (?).

7. Degeneration of brain stem (?).

#### *Histological Diagnosis*

Myeloma, mixed cell type, with metastasis to prostate, pancreas, stomach, kidneys, brain stem and pituitary.

## Woman's Auxiliary

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### CANCER PROJECT

At the convention of the Women's Auxiliary to the Arizona State Medical Association in June, 1944, a motion was made to endorse a project to further the educational program of the Arizona Society for the Control of Cancer. The first step toward organizing the state of Arizona in behalf of the Society for the Control of Cancer was made by Mrs. James H. Allen, president of the Women's Auxiliary to the Arizona State Medical Association. Mrs. Allen invited the Regional Commander of the American Society for the Control of Cancer to address a group of women at a luncheon held at Prescott, Arizona, Wednesday, September 6, 1944. At this meeting Mrs. Emily G. Bogert, the Regional Commander, gave an address, a review of which follows this report.

At the close of the address at a short business meeting Mrs. Thomas A. Hartgraves was elected State Commander of the Arizona Society for the Control of Cancer. The following Auxiliary members have been appointed on the organization board: First Vice Commander, Mrs. James H. Albeck, Phoenix; Mrs. Ernest A. Born, Northern len, Prescott; Second Vice Commander Mrs. L. D. District Commander, Prescott; Central District Commander, Mrs. Maurice Rosenthal, Phoenix, and Southern District Commander, Mrs. Edward M. Hayden, Tucson.

MRS. T. A. HARTGRAVES,  
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### REVIEW OF ADDRESS

Mrs. Emily G. Bogert of Denver, Colorado, Regional Commander of the American Cancer Soci-

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\**Laryngoscope*, Feb. 1935, Vol. XLV, No. 2, 149-154. *Laryngoscope*, Jan. 1937, Vol. XLVII, No. 1, 58-60. *Proc. Soc. Exp. Biol. and Med.*, 1934, 32, 241. *N. Y. State Journ. Med.*, Vol. 35, 6-1-35, No. 11, 590-592.

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ety, was in Arizona the week of September 6, 1944 in the interests of organizing a State Cancer Society and in planning for an inter-organization educational program in cancer control.

In presenting cancer as a major health problem in the United States, Mrs. Bogert stated that one out of every nine deaths in the United States is attributed to cancer—that each year in the United States 160,000 people die of cancer.

There are from 600,000 to 800,000 cases of cancer each year. Cancer is not a hopeless disease. Over 36,000 cures are on file with the American College of Surgeons. Only "late" cases of cancer become incurable because of neglect or delay or lack of proper ethical treatment. The cancer control program offers hope through education of symptoms which may lead to cancer and through stressing the importance of regular complete physical examinations.

Mrs. Bogert further stated "All of us who are wrestling with the problem of saving lives through educating the public are aware of the complexity and difficulty in the job we have undertaken. If through united interorganization cooperation we can arouse the public to an awareness of the precancerous period—if we can teach the public that these precancerous lesions may lead to cancer if not carefully observed, we shall go far."

The Cancer Control program is under the supervision of the American Cancer Society and has the approval of the American Medical Association and Public Health Service. Its program is based on science, application of science and education.

The slogan of the Society is "Early Cancer Is Curable. Fight it with Knowledge."

## Book Reviews

"POLIOMYELITIS." The Rejection of Neurotropic Streptococci to Epidemic and Experimental Poliomyelitis and Poliomyelitis Virus, Diagnostic Serologic Tests and Serum Treatment, by Edward C. Rosenow. M. D., Professor of Experimental Bacteriology, University of Minnesota, Mayo Foundation, Rochester, Minnesota. Published by The International Bulletin, Volume A-44, 319 West 103rd Street, New York City.

According to the Foreword, this volume of The International Bulletin is the first to be given over entirely to the work of one author. Presented here is the Monograph of Dr. E. C. Rosenow covering the twenty-seven years of research on the streptococcal etiology of poliomyelitis. In the Preface Dr. Rosenow says, "This monograph, I believe, summarizes my work and will meet the objections that have stood in the way of a general acceptance of the primary streptococcal causation of poliomyelitis and the streptococcal source of the virus."

The text deals in detail with the immense amount of work Dr. Rosenow has put into his subject, much of which has been hitherto unpublished. He described in detail techniques and results of his numerous experiments covering various phases of the problem such as:





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2. Finding of diplococci and streptococci in the lesions of the brain and spinal cord in epidemic and experimental poliomyelitis.

3. Microdiplococci in filtrates of poliomyelitis virus.

4. Specificity of the streptococcus as shown by inoculation of animals, cataphoresis, agglutination tests, and precipitin reactions.

5. Poliomyelitic streptococcal antigen in the serum of persons and monkeys during attacks of poliomyelitis.

6. Streptococcal cutaneous tests for susceptibility to poliomyelitis and a cutaneous test diagnostic of specific streptococcal infection in poliomyelitis and other diseases.

7. Precipitation and cutaneous reactions as measures of streptococcal antigen.

8. The protection of monkeys against experimental poliomyelitis with vaccine and antiserum prepared with the streptococcus, and the treatment of epidemic poliomyelitis with the poliomyelitic antistreptococcal serum.

9. Experimental production of a filtrable transmissible agent from neurotropic streptococci.

This partial list should give the reader some idea of the thoroughness of Dr. Rosenow's work and its presentation. Every physician or worker who has to deal with poliomyelitis should avail himself of this contribution to the growing store of knowledge concerning the disease. Those who are inclined to be skeptical owe it to themselves and to Dr. Rosenow to read this presentation.

R. L. F.

"METASTASES—Medical and Surgical," by Malford W. Thewlis, M. D., Attending Specialist in General Medicine: United States Public Health Hospitals, New York City; Attending Physician, South County Hospital, Wakefield, Rhode Island; Special Consultant, Rhode Island Department Public Health, Author Care of the Aged (Geriatrics), Preclinical Medicine.

Foreword by Hubert A. Royster, A.B., M.D., F.A.C.S., Honorary Chief Surgical Service, Rex Hospital; Chief-of-Staff, St. Agnes Hospital; Consulting Surgeon, Dix Hill State Hospital; Fellow American Board of Surgery, Raleigh.

Published by the Charlotte Medical Press, Charlotte, N. C. 230 pages, 13 illustrations. Price \$5.00.

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F. J. M.

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(Continued from page 273)

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